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POLIOMYELITIS AND MURRAY VALLEY ENCEPHALITIS: A COMPARISON OF TWO NEUROTROPIC VIRUS DISEASES.[1]

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THERE are only two important diseases in Australia which are known to be due to neurotropic viruses, poliomyelitis and the disease which we have recently named Murray Valley encephalitis (MVE). The two diseases present both similarities and differences and between them they probably exemplify all the important features of virus diseases of the central nervous system. Both provide for the epidemiologist problems of extraordinary interest, and for those who would try to prevent them, an extremely difficult task. I shall attempt tonight to discuss these two diseases more or less in parallel, with most of the emphasis on those aspects with which I am most familiar, the characteristics of the viruses, the way in which the viruses survive in Nature and produce human disease, and the possibilities of devising measures for their prevention.

I shall say nothing of the clinical problems of diagnosis or treatment beyond a brief and dogmatic statement that at the present time there is no known means of influencing either type of infection beneficially once the diagnosis can be made. The problems of the post-infection paralyses or other sequelæ are also completely out of my field.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on December 13, 1951.

Perhaps I might start by indicating the basic similarity of the two diseases. Both represent in their clinical manifestations an invasion of the central nervous system by the virus. In neither is this involvement a necessary result of infection. It is far commoner to suffer infection without any symptoms whatever. More than a quarter of Mildura's population have probably been infected by MVE virus, and I should be very surprised if anyone in this audience has escaped being infected with polio virus.

From the point of view of the epidemiologist endeavouring to understand and control the incidence of the two diseases, the main objective is to correlate the overt behaviour of the clinical disease and the more extensive invisible activity of subclinical infection, with what can be called the natural history of the virus concerned. A virus is just as much a species of living organism as a jellyfish or a rabbit. Through a complex process of food seeking, multiplication and movement to new sources of nutrition on the one hand and of all types of destruction by physical and biological agents on the other, the species manages to survive from year to year. It is now a truism that we cannot hope to understand the epidemiology of an infectious disease unless we can provide a satisfactory account of how the parasitic microorganism survives in Nature. This ecological approach to infectious disease seems to me to be particularly important in the two diseases we are considering and most of this lecture is concerned in one way or another with this type of approach.

Perhaps the most obvious way to look at poliomyelitis as a virus disease is to concentrate on the essential lesion in the anterior horn cells. The virus reaches these cells, multiplies freely within them and in the process destroys their functional activity completely and permanently. If one injects poliomyelitis virus into the central nervous

system of a monkey this is a reasonable sketch of what happens, and in fact the history of research and understanding in poliomyelitis since experimental work began in 1909 is very much the story of the progressive modification of this simple picture.

The acutely fatal case of poliomyelitis, where *post mortem* we find the cord saturated with poliomyelitis virus and most of the anterior horn cells necrotic, is the rare limiting case of a very wide range of interactions between the two organisms, man and the poliomyelitis virus. Experimental virus work is becoming more sophisticated than it was and it is fair to state that there is at present a strong reversion from the old tacitly accepted picture that the usual result of interaction between virus and cell was multiplication of the virus with necrosis and disintegration of the cell.

For technical reasons academic work on the interaction of virus and cell has been done with viruses other than those of poliomyelitis and encephalitis and I shall make only passing reference to the large amount of fundamental work on influenza virus that has been reported in the last three or four years. All I wish to mention is the many ways in which the process of infection can be blocked or modified even when we are dealing with viruses of artificially enhanced virulence and the highly susceptible cells of the chick embryo. If dead or partially inactivated virus is inoculated with highly virulent virus, infection by the latter may be inhibited by a process of interference. Many strains of virus appear to contain rather large numbers of units which are much more prone to interfere than to infect. Rather closely related is the recent finding that under some conditions viruses do not reproduce themselves but produce what we call "incomplete virus" particles which have many of the properties of virus but cannot initiate infection.

Findings of this sort will probably sooner or later be extended to other viruses, but even now they seem to throw some light on the central feature of the two diseases we are considering, the enormously greater number of non-symptomatic than of symptomatic infections. When a virus infection is initiated in the body of a person with no preexisting immunity, it is only on the rarest occasions that the virus finds it possible to sweep through and produce serious damage in all or most of the cells that are theoretically susceptible to its action. Much more often the virus has to fight its way through a whole series of obstacles that we are only beginning to particularize —obstacles at the surface of the cell and within the cell, interference phenomena by which a non-virulent virus can block the activity of a virulent one, failure of cells in which virus has multiplied to liberate the new brood of virus particles and so on. None of these processes have yet been clearly defined, but they must be invoked if we are to understand the basis of subclinical infection.

Perhaps I should make it a little clearer as to what we understand by subclinical infection in virus diseases. In general there are only two ways by which we can prove that a person is infected by a given virus. The first is to show that at the relevant time virus is present in tissues or secretions in amount sufficient to make it certain that the virus had multiplied in the person's tissues and was not merely a casual contaminant. The second is to demonstrate that an immunological change in serum antibody or skin reactivity has appeared shortly after the time of presumed infection. In poliomyelitis studies we may cite Gear's report that when he tested the fæces of nine young Negro children in South Africa he found three to be excreting poliomyelitis virus without showing any signs of infection. From the other aspect we might mention Paul and Melnick's findings of Lansing type antibodies in an Eskimo community which nineteen years previously had had two cases of paralytic poliomyelitis. Nearly everyone twenty years of age or older showed antibody, none of those who were under that age. Anderson and Donnelley's results with MVE complement fixation tests show that a random sample of the population of the Mildura region gives about 20% of reactors. We may have to postulate 500 subclinical infections for each case of frank encephalitis.

It is possible that subclinical infection may occur without provoking a detectable antibody response and sometimes it may be necessary to postulate such an occurrence to account for certain epidemiological happenings. In this discussion, however, I shall use the term subclinical infection as indicative of a condition in which, if appropriate technical means were applied, virus multiplication could be demonstrated and a subsequent appearance of antibody detected. Everything points to this type of subclinical infection being responsible for the main epidemiological features of poliomyelitis and probably for the mosquito-borne encephalitides as well.

The broad thesis I wish to present for both diseases can be stated then as follows: Following primary infection with the virus, infection is initiated which in rare individuals may extend to and seriously involve cells of the central nervous system. In most persons multiplication and spread is limited by a series of obstacles not yet well understood, and little or no symptoms result. Sufficient multiplication of the virus, however, occurs to provoke an immunological response. The ratio of clinical to subclinical attacks varies with the strain of virus concerned. A subclinical infection by a low grade virus will in general protect against subsequent symptomatic infection by a more virulent strain.

The Problems of Poliomyelitis.

In any short general discussion of poliomyelitis it is necessary to have the courage to provide a summary of what one regards as the relevant facts that have been established from forty years' research on the disease. I think that many workers would consider that what I am going to say is an over-simplification of the position, but I can assure you that it is not said without long consideration and much talk with many of the leading workers in America and England.

It is not so many years since, in the course of a general discussion on virus diseases, one could say that although almost every other part of the body was subject to one or other virus infection, none were known to involve the intestinal tract. Things have changed greatly and now we know that the intestine is the site of election for three poliomyelitis viruses, at least ten Coxsackie-type viruses and almost certainly the virus of infectious hepatitis. The natural habitat of the poliomyelitis viruses is in the lining of the alimentary tract, the small intestine and the tonsillar region being the preferred sites of multiplication. Enormous amounts of poliomyelitis virus are excreted in the fæces of any community in which poliomyelitis is active, so much so that it can be isolated from a few cubic centimetres of mixed city sewage. There is a growing belief that poliomyelitis is spread predominantly by transfer of infected fæces in one fashion or other. Droplet infection from pharyngeal sources cannot be excluded and is still favoured as the important mode of spread by some authorities.

Eventually it may be decided that the poliomyelitis viruses and the Coxsackie viruses represent a single interrelated group of viruses, all of the same very small size and with a similar localization in the body and means of spread. For the present, poliomyelitis viruses are defined by the additional quality of ability to provoke the characteristic anterior horn-cell lesions in man or on intracerebral inoculation into one of the susceptible species of monkey. There are three immunological types known by the names of type strains as Lansing, Brunhilde and Leon. One of these, Brunhilde, is, in the United States at least, of vastly more importance than the others as a cause of the paralytic disease. Only a single strain of type Leon is known and I shall make no further mention of it. Lansing is only a rare cause of clinical poliomyelitis. It shows hardly any serological relationship to Brunhilde and in addition differs importantly in its capacity for adaptation to rodent hosts, cotton rats and mice. This makes it much easier to study than the Brunhilde strains, which are pathogenic only for monkeys. Within either main group of poliomyelitis viruses there are individual variations in serological detail and in the humanly important character that we can term invasiveness. I am not

going to touch on the processes by which a poliomyelitis virus can pass from the bowel or throat to the central nervous system, but we do know that there are certain regularities between the presence of poliomyelitis virus in the intestinal tract and the occurrence of the paralytic disease. Both host and virus factors are involved in this relationship. On the side of the virus we know that there are differences between strains in their capacity to infect the central nervous system which we can call their invasiveness. I have tried to represent this in a diagram showing the sort of incidence of paralytic poliomyelitis one might expect with four strains of each type in a non-immune population. Differences in the invasiveness of different strains are almost certainly the main reason for the differences in intensity of poliomyelitis epidemics in any community which has maintained a reasonably constant social structure.

Disease is always a matter of interaction between the parasite and the host and nowhere is this more evident than in poliomyelitis. On the side of the host, by far the most important factor is the age at which infection first

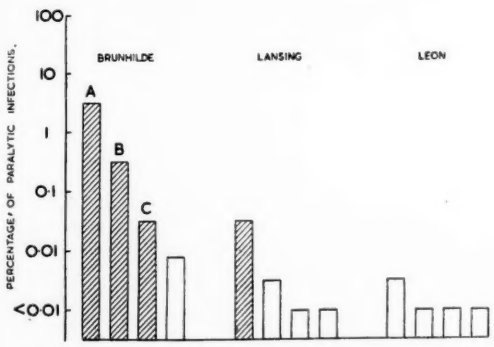

FIGURE I.

A diagram to indicate the percentage of paralytic infections which might be expected to follow infection of the alimentary canal with strains of the three poliomyelitis virus types. Shaded bars represent strains that would be expected to produce some recognizable clinical cases. Logarithmic scale. A, B and C identify the strains on which Figure II is based.

occurs. Nearly all the difficulty of understanding the epidemiology of poliomyelitis disappears if one accepts the view that the proportion of individuals infected for the first time who suffer from paralytic infection varies characteristically with age, being lowest in infancy and reaching a maximum in adolescence and young adult life. Another diagram will perhaps serve to make this clear. It must be stressed that here we are dealing with a circumstance that never exists in any large centre of population, namely, a wholly non-immune group of persons. Such can be found only in small isolated communities. A virgin soil outbreak of poliomyelitis has just the character shown by one of the curves in the diagram with the main incidence of paralysis on adolescents and young adults. Some of the places where poliomyelitis has shown this character are St. Helena, Guam, New Guinea. Solomon Islands, and two isolated Eskimo villages in the Canadian Arctic. These virgin soil outbreaks are dramatic occurrences which could hardly escape record in the accounts of early European travels and colonizations if they occurred. It is therefore extremely interesting that there is to my knowledge no account of such an outbreak earlier than that which occurred in 1899 in Guam.

The importance of limiting such discussion to primary infection is of course due to the fact that primary infection, whether clinical or subclinical, leaves behind it a significantly altered bodily condition shown by some degree of immunity. This immunity can be experimentally demonstrated in chimpanzees in two ways. If a previously uninfected ape is fed the virus and daily tests are made on the fæces, it is found that virus appears in two to four days and persists for two to three weeks. A second feeding months later with the same strain of virus is followed by no excretion after the actual inoculum has been disposed of. This acquired resistance to intestinal implantation of the virus is the first manifestation of immunity. The second is the appearance in the blood of virus-neutralizing antibody. We can feel quite confident that in addition the development of immunity also greatly diminishes the chance that virus can pass from the alimentary tract site to the central nervous system. There is as yet no experimental evidence for this, but there is just no other conceivable explanation of the epidemiological facts. The clearest way to exemplify this is, I think, to compare the age incidence of diagnosed poliomyelitis in two contrasting island epidemics—Malta in 1943, St. Helena in 1947.

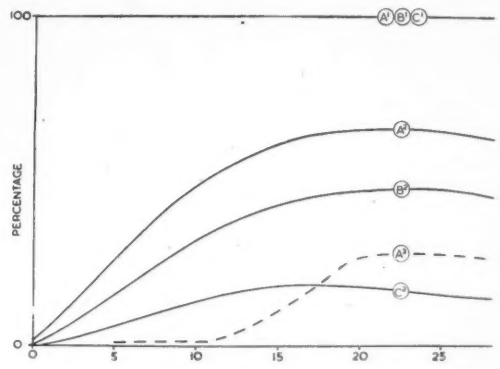

FIGURE II.

A diagram of the effect of three strains of Brunhilde virus on a hypothetical completely non-immune population of all ages. A^1, B^1, C^1 indicates that 100% infection of all ages is postulated, A^2, B^2, C^2 the percentage of paralytic infection at the ages shown, while A^3 suggests a probable age distribution of fatal cases with strain A.

In the graph (Figure III) the incidence shown in five-year groups is in terms of the total population of the island concerned. Although the age distribution of the populations at the time is not available, we can probably assume that both were reasonably similar. It will be seen that the incidence under five years is approximately equal, but that on Malta this age group contains almost the whole of the victims, while on St. Helena it represents only a small fraction. Put very broadly, infants on the two islands were similarly affected, on St. Helena older children, adolescents and young adults were heavily involved, but on Malta clinical illness hardly touched these groups. The only adult cases observed in the Maltese epidemic were in British servicemen.

Actually the intensity of attack on the younger ages in Malta was more striking than is shown in the diagram. The actual figures for the first five years of life were 0-1, 69; 1-2, 114; 2-3, 109; 3-4, 72; 4-5, 33. From three years onward, then, some factor was in action in Malta that was absent in St. Helena. That factor can only have been the subclinical infection of the population with poliomyelitis virus in previous years.

There is, in fact, evidence that small numbers of poliomyelitis cases had occurred in each of the previous ten years, averaging 2·6 *per annum* and almost wholly in infants. We can be quite certain that these few cases were the visible part of a process of infection that involved subclinically almost the whole of the infant population. What work has been done on the isolation of virus and the study of antibody at different ages in communities of low economic standards fully confirms this interpretation.

We are then forced to the conclusion that the school-age children of Malta in 1943 were protected against a poliomyelitis epidemic due to a strain or strains of the highest

invasiveness by the fact of repeated subclinical infection during their earlier years by poliomyelitis strains of low invasiveness. I know of no facts to contradict the dual deductions (*a*) that the prevalence of poliomyelitis in advanced rather than in backward countries is due primarily to the relative absence of subclinical immunization in the earliest years of life, and (*b*) that the only logical approach to the prevention of poliomyelitis is the development of some means of artificial immunization which will effectively replace the natural process.

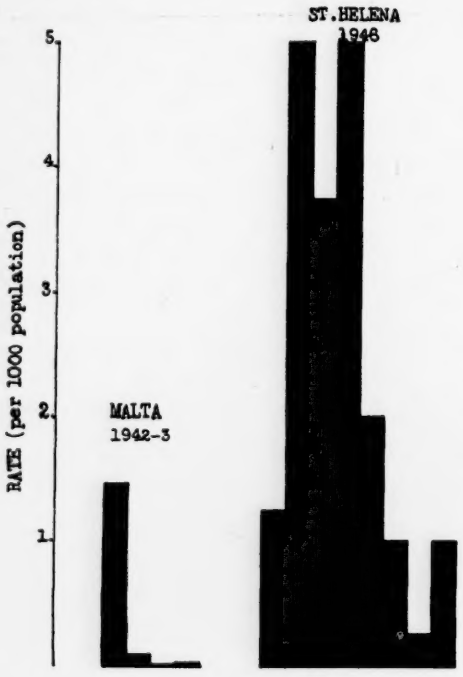

FIGURE III.

The age incidence of poliomyelitis in two island epidemics: Malta, 1942-1943; St. Helena, 1946. Number of cases per 1000 total population in five-year age groups. Figures for Malta from Seddon *et alii* (1945), for St. Helena from Nissen (1947).

I have not time in this lecture to develop the theme that advances in living standards, cleanliness and persona hygiene by diminishing the transfer of fæcal material has delayed the average time of first infection with poliomyelitis and greatly increased the incidence of visible paralytic disease. I am quite certain that one can read the history of poliomyelitis much more intelligently on that working hypothesis than on any other. The appearance of the disease almost strictly in accord with the successive rises in living standards in different countries, the changing age incidence and the way in which its rising or near-stable incidence contrasts with the falling incidence of virtually every other infectious disease of childhood—all these then fall into line.

What are the prospects of immunization? There are technical reasons why a conventional killed vaccine is a very improbable solution, the most important being the physical impossibility of obtaining sufficient actual virus antigen. The only visible approach is the use of a living non-virulent virus administered by the natural alimentary route of infection. I have given reasons for thinking that the average poliomyelitis virus strain that produces a few cases only during its passage over a large community of children, will produce virtually no paralytic infections in infants. Perhaps a strain could be chosen which in such infants could be relied on completely for its innocuousness and for its power to immunize. If such a strain could be grown on a large scale in tissue culture by Enders's methods, it would, I think, rapidly become a widely or universally used prophylactic.

It would be foolish to minimize the enormous difficulties in developing an effective practical means of large-scale immunization against poliomyelitis. When we realize that to be effective it must be applied in the first year or two of life and that there is no certain means of ensuring the harmlessness of a living poliomyelitis virus except actual test in infants, we can appreciate the courage (or foolhardiness) that will be needed. It may be that no Australian community will tolerate the experimental development of any such method. Nevertheless, I would repeat that unless our reasoning is badly at fault every increase in the amenities of civilization will render the community progressively more subject to epidemics of poliomyelitis. At some point the demand for protection will increase to the point at which the lesser danger of paralysis from the immunizing procedure may become acceptable as the price that must be paid for protection of the majority.

Murray Valley Encephalitis.

Early in February this year news was received that there were several severe cases of encephalitis in the Mildura and Mooroopna base hospitals in northern Victoria. Dr. S. G. Anderson at once visited Mooroopna and found five infected persons in hospital; one patient had just died and at a post-mortem examination samples of brain were removed aseptically and brought to Melbourne packed in dry ice. Brain emulsions were inoculated into a variety of animals and into chick embryos by several routes. No results were evident except in the chick embryos inoculated on the chorio-allantois. Two or three small white foci were visible on the membranes and a second passage in the same situation gave quite definite virus foci. By February 23 it was known that a virus capable of infecting adult mice intracerebrally and baby mice by peritoneal inoculation as well as producing chorio-allantoic foci and death in chick embryos had been isolated and was the presumptive cause of the disease. Since then the situation has been intensively investigated by my Victorian colleagues both inside and outside of the Hall Institute (S. G. Anderson, 1952; E. L. French, 1952; E. G. Robertson and H. McLorinan, 1952; E. G. Robertson, 1952; S. G. Anderson *et alii*, 1952), and through the courtesy of Dr. Anderson, Mr. French and their collaborators I can present their main results this evening.

Although perhaps it is not strictly relevant to the scientific problems of the disease, I think I should give you a little of the background of public interest against which the investigations were conducted. The encephalitis cases occurred just at a time when the much-publicized myxomatosis epizootic in rabbits became well established along the Murray River and there was a very rapid and understandable growth of public suspicion that myxomatosis virus was responsible for the human disease. It therefore became a matter of nearly first-rate political importance to exonerate the myxoma virus. This gave a certain urgency to the development of serological methods by which it could be shown with certainty that the cases of encephalitis occurring in northern Victoria were all due to the newly isolated virus and not to myxomatosis virus. The work has perhaps received rather more publicity than its importance warrants, but I think the justification of that publicity has been its effect in stimulating active public cooperation, especially in the Mildura area. Field epidemiology can be effective only when the communities concerned are actively interested in their own health problems.

The disease was named Murray Valley encephalitis as an outcome of a discussion between Dr. Anderson and myself. Most such diseases have been given a geographical designation and we had also in mind the high probability that the "X" disease epidemics of 1917-1918 represented the same type of infection. These involved an extensive portion of the Murray-Darling basin. A perusal of the clinical, epidemiological and experimental papers written

on the "X" disease outbreaks (J. B. Cleland *et alii*, 1917) provides no evidence against the identity of the disease with that of last summer.

A full clinical account of the disease has recently been published by Graeme Robertson and McLorinan (1952) and here I shall only say that in typical cases the classical symptoms of acute encephalitis are presented—a fairly sudden onset with headache, vomiting and high fever, rapid deterioration of consciousness usually passing into coma with variable disorders of muscular function. In 17 of the 40 cases in which a diagnosis was made the patients died. Some patients made a complete recovery, but others had residual physical or mental damage. Most patients were treated with all available antibiotic drugs without evidence of any beneficial effect.

The epidemiological features of the 1951 epidemic may be summarized in a map showing the geographical distribution of the cases (Figure IV) and in two tables in

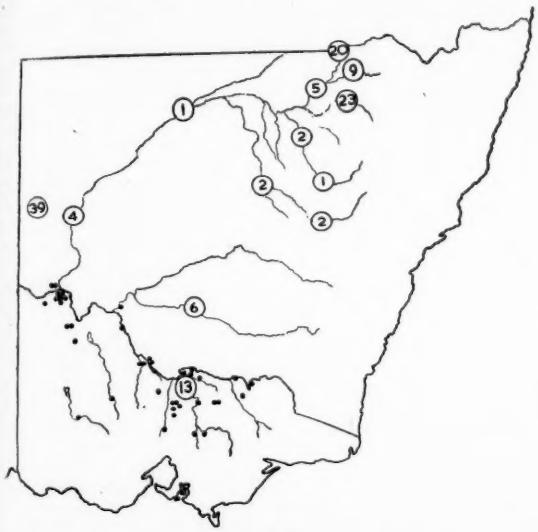

FIGURE IV.

Map of New South Wales and Victoria to show distribution of diagnosed cases of Murray Valley encephalitis, 1951 (black dots), and of X-disease in 1917-1918 (circled figures), from Cleland *et alii* (1918). Note association with the river systems of the Murray basin.

which the incidence by months and the age incidence are presented both for the "X" disease epidemics of 1917 and 1918 and for the present outbreak.

The geographical data are virtually limited to Victoria and show a concentration of cases in the centres of population along the Murray River. No cases have been observed in which infection must have been contracted south of the Dividing Range.

The monthly incidence is similar for each epidemic, cases being almost confined to the hot months of January, February and March.

In all three epidemics a majority of the cases occurred in children, but all other ages were also represented.

The 40 cases of the 1951 outbreak must be regarded only as a very small visible fraction of the whole extent of human infection by the virus. Nevertheless the distribution of cases in time and place probably presents a fairly accurate reflex of the whole epidemiological process. Even without any further information such a distribution would point strongly to an infection carried by some freely moving insect vector from a non-human reservoir of infection. This was in fact a conclusion I reached in regard to "X" disease in 1934 (Burnet, 1934), although the suggestion then made that sheep represented the probable reservoir was almost certainly wrong.

Much more information is, however, obtainable by the method of serological survey which nowadays is the mainstay of most epidemiological work. Recent infection by the virus in human beings, animals or birds leaves evidence in the form of specific antibody in the serum. This can be detected either by a complement fixation test which requires only test-tube manipulations, or a neutralization test in mice which is a more expensive procedure. For technical reasons the complement fixation test has been used with mammalian sera and the neutralization test with bird sera.

The important results of such surveys have been as follows:

1. A relatively large proportion of persons in Murray Valley centres showed evidence of recent infection. Early studies were directed principally towards contacts of clinical cases and persons giving a history of minor illnesses that possibly represented non-encephalitic infection by the virus. The incidence of antibody in the two groups was very similar and approximately the same as in a random sample of persons from the same district. In the Mildura district the level of positive sera was 20% in adults, 7·0% in children; in the Shepparton district the levels were 4·5% and 4·7% respectively; while in southern Victoria there was a level of less than 1·0%.

2. In such an endemic region as Mildura there is a high incidence of positive sera from horses and dogs (over 50% in a small series) and again no positive reaction from animals of the same species south of the Divide. Sheep and cattle failed to provide positive results.

3. A high proportion of some species of native water-birds, herons, cormorants, swans and duck, show neutralizing antibody.

4. In a small series a high proportion of domestic fowls (seven out of nine) were found to possess circulating antibody.

In any attempt to interpret these findings, we must first make the assumption that the antibody present is specific evidence of past infection by the virus of Murray Valley encephalitis. The justification for this is first that in 15 cases of encephalitis occurring in the northern part of Victoria and diagnosed clinically as Murray Valley encephalitis, the patients survived long enough to allow a "second" sample of serum to be obtained and tested. All but one gave a definite positive reaction. The second negative criterion of specificity is that human and equine sera from non-endemic regions contain no antibody. A very important gap in our knowledge is our ignorance of how long antibody remains detectable after infection. In the absence of such data it is difficult to draw any deductions from, for example, the observed 20% incidence of antibody in Mildura. However, since we know that complement fixing antibody titres in most virus infections usually fall rather rapidly, we shall probably be more nearly correct if we assume that the antibody recorded in 1951 is predominantly a result of infection during the preceding summer.

A situation in which human beings, horses, domestic fowls and a variety of water-birds over a wide area of country all show evidence of 20% to 50% infection has only one reasonable epidemiological interpretation, namely, that the infection is spread by a common highly efficient and freely mobile insect vector. Taken along with what is known of other virus encephalitides, the vector can only be one or other species of mosquito. We have not the slightest doubt that adequate investigation will show that the disease is in fact mosquito-borne.

It is premature to go further until such investigations can be carried out, but even at this stage I think it may be advisable to construct a generalized picture of the natural history of the disease. We must have some form of working hypothesis on which investigations may be planned or emergency measures devised.

The following outline, then, is merely such a working hypothesis to be modified or abandoned as the facts are obtained. We believe that the major process leading to an epidemic takes place in summer in environments where large numbers of new season birds, nestlings and young

adults, are exposed freely to the bites of an effective vector mosquito. Such environments are common along the Murray where a great variety of water-birds nest, often in very large localized rookeries. Once an infection is initiated in a young non-immune bird, mosquitoes will be infected during the phase of viræmia and pass the virus to other susceptibles. Under suitable conditions this would lead fairly rapidly to the infection of the great majority of birds in the area. It is probable that the infection of young wild birds produces little or no overt illness, merely a transient viræmia lasting a day or two which provides both an opportunity for infection of further mosquitoes and a stimulus for the production of a firm immunity against reinfection. In a relatively stable bird-mosquito association we should expect a sharp subclinical epidemic lasting at most a month or two and disappearing as an increasing proportion of the birds became immune. This may be regarded as the primary epidemiological process. Two questions immediately arise. The first and more fundamental is, how does the infection persist from one season to the next? The second is, how do human infections occur?

The answers to these two questions can obviously be obtained only by close study of field conditions, but we can make some cautious deductions from the evidence already available. The matter of human infection can be dismissed fairly readily. It is reasonable to believe that any mosquito in which the virus can undergo its vector cycle and which will bite man, may be able to initiate human infection. The mosquito must first feed on some bird or mammal which at the time has a high concentration of virus circulating in its blood; then after the appropriate period for virus multiplication and passage to the salivary glands, human infection can be produced. The investigations that will be required this summer then are first to determine which mosquitoes prevalent in the epidemic areas are naturally infected or capable of experimental infection, and second, to find which birds and animals develop after infection a high enough virus content in the blood to infect mosquitoes. With that background it should be possible to build up a satisfactory picture of the natural history of the disease as it affects the human population.

The means by which the virus survives from year to year presents a fascinating but much more difficult problem and one not specially relevant to the immediate public health objective. There are two main possibilities; the first is that transfer of virus between birds goes on all the year, a variety of mosquitoes being involved. In the colder months passage is greatly slowed down, but it is at least conceivable that sufficient concentrations of vectors and susceptible hosts remain available somewhere in the eastern Australian region to ensure the maintenance of a trickle of infection throughout the year.

The second hypothesis based on American work is that the virus survives in some ectoparasite of birds, most likely a mite (Dermanyssus or Liponyssus) and is perhaps transmitted from one generation to another by transovarial infection. Such mites could continue to feed on immune birds and in the following nesting season transfer the virus to nestlings from which mosquito transmission would reinitiate the main cycle.

To differentiate between these two hypotheses or to establish some other alternative will obviously require both intensive work and good fortune. The chances of success seem in fact so small that I doubt whether the problem should be given more than a low priority in the investigational programme.

Something must be said about the possibilities of preventing or minimizing future outbreaks of this disease. There are two possible approaches: we may either try to prevent infection of human beings by appropriate action against either reservoir or vector, or by the use of some means of immunization render infection harmless when it occurs.

The first is obviously the more desirable approach, and we have the experience of work on malaria and yellow fever in the past to help us in devising means of interfering successfully in the cycle by which the virus survives in Nature. Until the details of the vertebrate reservoirs and mosquito vectors have been worked out it is impossible to say where, if anywhere, is the weak link in the chain that can be broken by public health action. There may be some relatively simple way of virtually eliminating the possibility of human disease or on the other hand it might become clear that the problem is quite insoluble by any practicable means. The essential need at the present is a close study of the conditions in the field during the epidemic period. Such work is now in progress, and we are hopeful that by the end of the summer a great deal more will be known. Until we have the necessary information it would be wholly unjustifiable to advocate any large-scale anti-mosquito measures. We can do little more at this stage than to advise that children be protected as far as possible from mosquito bite during the months of January to March.

Theoretically some form of immunization might provide a sufficient increase in immunity to reduce the incidence of encephalitis in those infected from about 1:500 to 1:10,000 or less. The only large-scale use of such a measure on record is the immunization of military personnel against Japanese B encephalitis. No satisfactory assessment of the value of this immunization has yet been published, and in a situation where many hundreds must be immunized for every one protected, we have to be particularly sure that no harm whatever can be done by the vaccine. Most virologists are still unhappy about the use of virus vaccines containing a large proportion of host tissue, especially if nervous tissue is included, and immunization against Murray Valley encephalitis should probably not be considered at the present time.

There is one final and very important point to be made. Money and effort will be spent in public health work only if the benefit to be gained is regarded by the community as commensurate with the expense. If Murray Valley encephalitis should continue to produce considerable numbers of victims in future years, an effort to prevent the disease at least in the main centres involved will be demanded. On the other hand we may find in the future, as in the past, that only on rare occasions when some unusual combination of ecological circumstance arises does the virus spill over from its avian reservoir into the human population. In the intervening years it survives amongst the wild birds, but makes no human impact whatever. If this is the case we shall simply have to adopt the realist attitude that encephalitis is just one of the inescapable risks of Australian life (like bush fires, motor cars, and staphylococcal food-poisoning) against which specific measures cannot be effectively used.

Summary.

To summarize this presentation of our two significant virus diseases of the central nervous system in Australia:

Both poliomyelitis and Murray Valley encephalitis represent the visible result of infections which affect vastly greater numbers of people without producing any sign of serious illness.

Poliomyelitis spreads through the whole populated area by transfer from one human being to another, especially in childhood. Murray Valley encephalitis is transferred possibly in more than one step by mosquitoes, the infection being maintained in a reservoir of wild birds. The affected region is determined by those conditions which affect the number and activity of susceptible birds and the mosquito species capable of carrying the virus.

There is no visible means of preventing the movement of poliomyelitis virus in the community and hope of prevention in the future rests on the development of an effective immunizing agent. Present indications are that it will be necessary to use a living attenuated virus given by the alimentary route in infancy.

The significance of Murray Valley encephalitis as a public health problem in Australia has not yet been adequately assessed. Until investigations are more advanced it is not possible to say whether it will be practicable to use

anti-mosquito measures for the control of any future outbreaks that may occur.

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THE MORTALITY IN AUSTRALIA FROM ACUTE INFECTIVE DISEASE.

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THE mortality from certain acute infectious diseases is considered here as part of a general survey of the official mortality statistics in Australia over the years 1908 to 1945. Information dealing with deaths from infectious diseases has been abstracted from *Demography*, the annual bulletin of the Bureau of Census and Statistics, Canberra, with the aid of the bureau.

The Acute Infective Diseases.

In this paper the acute infective diseases will include most of the diseases of Class I of the "International List of Causes of Death" (fifth revision) together with certain other diseases from other classes of the International List. The diseases of Class I excluded from consideration here are tuberculosis, the venereal diseases, "purulent infections and septicæmia", and Hodgkin's disease. Diseases from other classes of the International List included here are non-epidemic encephalitis and non-meningococcal meningitis, which were classed at the fifth revision of the list among the diseases of the nervous system. Earlier revisions had also put non-epidemic meningococcal meningitis into the diseases of the nervous system. These conditions also are included here. The International List refers deaths from diarrhœa and enteritis to the diseases of the digestive system, but it seems better to include them with the other infective diseases, as is done in this paper. However, it is not practicable to include infective diseases of the respiratory system, and it will be more convenient to deal with them as a whole at a later date, although I have followed the International List in dealing with influenza here. The group of diseases so defined I shall refer to as "acute infective disease" in this and the following paper, to avoid unnecessary verbiage. It will be seen that the group is responsible for a considerable proportion of the deaths in childhood and is of great importance in the study of the decline in the mortality rates, both in childhood and at other ages.

The Importance of the Acute Infective Diseases as a Cause of Mortality in Australia.

For either sex and for each age group the number of deaths from acute infective disease has been expressed in Table I as a percentage of deaths from all causes. This has been done for five different periods in the years 1908 to 1945. When attention is fixed on any given period, the relative importance of the group is greatest among children aged one to four years, rather less in the first year of life and at school ages. From Table I the group appears to have less relative importance in adult life, especially in the most recent period, 1941 to 1945. However, in earlier years, such as the period from 1908 to 1910, the group still was an important cause of death in adult life. Meningitis, influenza, gastro-enteritis and the typhoid fevers were still causing a considerable proportion of the deaths in adult life. Influenza was more important in the next decade, 1911 to 1920, owing largely to the influenza pandemic of 1919. In the more recent periods these four diseases—meningitis, influenza, gastro-enteritis and typhoid fever—have been responsible for fewer adult deaths. In Table I, when the experience of any age group for either sex is traced, the decline in the relative importance of the acute infective diseases may be noted. In other words, changes in hygiene, treatment and other more indefinite environmental factors have been more effective against acute infective disease than against disease in general and against other causes of death, such as violence. More precise measures of these changes in mortality are the death rates, which are dealt with in the following section.

The Death Rates from Acute Infective Diseases in Australia.

In Table II are set out the death rates from acute infective diseases in Australia as deaths *per annum* per million mean population of persons of the same age and sex. These age groups in Table II correspond with the groups usually used in standardization. The groupings are rather broad for some purposes, such as a study of the mortality in childhood, and so an additional table, Table III, gives the mortality for finer age groupings in childhood.

The most striking improvements have taken place in the first year of life, for at this age the death rates have fallen to one-seventh of their original values in less than forty years. However, improvements have taken place at all ages. In a later section the effects of this fall in the death rates on the crude and standardized death rates will be considered in detail.

The death rates may be studied by age in any period, either in Tables II and III or in Figure I. The mortality rate is highest in the first year of life. It declines, at first rapidly and then more slowly, and is least around the age of twenty-five years for either sex. The mortality rate then tends to increase throughout adult life until in old age—that is, in the group of seventy-five years and over—the mortality rate is almost as high as in the first year of life. The reasons for this age distribution of the deaths are better appreciated by examining the death rates from individual diseases in later tables; but an idea of the age distribution of the individual diseases can be obtained from Figure I.

The trend of the mortality from acute infective disease is shown in Figure II, in which the mortality rate of each age group has been plotted against the calendar year. It is evident that there have been sharp declines at all ages. The graph of the mortality rate for males or for females, aged twenty-five to thirty-four years, shows an irregularity for the period 1911 to 1920, due to the influenza deaths in 1919. The death rates of males of military age in 1941 to 1945 may be spuriously low, owing to statistical practices in regard to the registration of deaths and to the estimation of the populations at risk in wartime (Lancaster, 1951).

The Contribution of the Acute Infective Diseases to the Decline in the Death Rates (All Causes) as Measured by the Crude and Standardized Death Rates.

For comparability with my other papers on Australian mortality experience, the crude and standardized death rates are given in Table IV. A notable decline in the death rates from acute infective diseases is apparent. This is first to be noted in the case of the crude death rates—

TABLE I.

The Importance of Acute Infective Diseases as a Cause of Death in Australia.

Period.	Sex.	Percentage of All Deaths due to this Group of Causes at Ages (Years) Given.								
		0	1 to 4	0 to 4	5 to 9	10 to 14	15 to 44	45 to 74	75 and Over	All Ages
1908 to 1910 ..	M.	35·01	50·48	38·54	44·54	20·13	11·70	4·88	4·64	14·98
1911 to 1920 ..	M.	30·97	51·42	36·20	37·35	21·46	16·50	7·96	3·53	14·73
1921 to 1930 ..	M.	23·54	45·63	29·02	26·66	14·58	6·43	2·88	2·65	8·58
1931 to 1940 ..	M.	11·78	37·90	18·26	36·64	12·28	4·94	1·93	1·58	4·36
1941 to 1945 ..	M.	10·53	34·45	15·62	26·50	13·17	4·27	1·29	0·93	3·15
1908 to 1910 ..	F.	37·31	50·48	40·63	39·48	24·41	8·23	5·10	5·61	16·12
1911 to 1920 ..	F.	32·32	53·01	38·34	45·23	26·75	13·03	5·99	4·50	16·11
1921 to 1930 ..	F.	24·41	47·34	30·52	31·17	16·93	5·92	3·32	3·05	9·24
1931 to 1940 ..	F.	13·16	40·62	20·55	32·26	15·78	4·42	2·16	1·81	4·72
1941 to 1945 ..	F.	10·99	38·00	17·01	27·02	14·71	3·90	1·35	1·22	3·24

that is, the deaths *per annum* per million of the total population. But there have been great changes in the age constitution of the Australian population, so that it is preferable to use a standardized rate as a measure of the mortality of the whole population. The rates have, therefore, been standardized onto three populations—firstly, the population of England and Wales, as enumerated at the census in 1901, then onto a life table population constructed from the data of the Australian census of 1933, and finally onto an artificial population of persons under the age of sixty-five years. Standardization onto this last population is equivalent to taking the mean of the mortality rates for each of the first thirteen of the quinquennial age groups.

It is of interest to compare the trend of the crude and standardized death rates from the infective diseases with the corresponding trends of the mortality rates from all causes—that is, to compare Table IV of this paper with Table IX of a previous paper (Lancaster, 1951), using rates per thousand *per annum*. Firstly, when a comparison is made with the crude rates, it is noted that the fall in the mortality rate between the period 1908 to 1910 and the period 1941 to 1945 due to the acute infective diseases is greater than the total fall in mortality. This is due to the aging of the population, which causes greater weight to be given to the degenerative diseases of old age in the more recent periods. More accurate comparisons can be made by reference to the standardized rates. Thus, with the rates standardized onto the populations enumerated at the census of 1901 in England and Wales, there were falls in the mortality from all causes of 2·6 per thousand for males and 3·3 per thousand *per annum* for females. The corresponding falls in the mortality from infective diseases were 1·4 for males and 1·1 for females per thousand *per annum*. So that over one-half of the fall in the male rate and about a third of the fall in the female rate can be ascribed to reduction in the mortality from the class of acute infective diseases. Similar comparisons have been made for the other standardized rates in Table V. It may be concluded that the reduction in the mortality from infective disease has contributed materially to the general fall in the death rates that has occurred in Australia over the years 1908 to 1945.

The Masculinity of the Mortality Rates from Acute Infective Disease

The influence of sex on the mortality from the acute infective diseases is indicated in Table VI, the masculinity being computed from the rates for the years 1931 to 1940. The masculinity has been defined as the ratio of the male death rate to the female death rate expressed as a percentage, or in other words, one hundred times the male death rate divided by the corresponding female rate. The masculinity in the first year of life for these diseases as a whole was high—113. At age one to four years it was 107; at ages five to nine years, 107; at ages ten to fourteen years, 114; and for all ages combined, 117. For several diseases in the group there is a ready explanation of such differences between the sexes. In the case of the intestinal infections, typhoid and paratyphoid fevers and gastro-enteritis and diarrhœa, there is probably an increased occupational hazard for males, for many men work under very primitive conditions in the outback and in isolated mining communities. No such explanations are available for the mortality in the first year of life. At this age the masculinity for the death rates from gastro-enteritis and diarrhœa is 130, and for the rates from influenza, 118. An exception to the rule is pertussis, which has a low masculinity of 81 in the first year of life, and 67 at ages one to four years. The bearing of this observation on the mortality from bronchiectasis will be discussed in a later section.

Some General Epidemiological Considerations of the Incidence of Infective Diseases in Children.

It is well known that mortality rates in general have tended to fall over the last fifty years, and that the decline has been most evident at the earlier ages and has been due in large part to the infective diseases. The explanations usually given emphasize the effects of sulphonamide drugs, the antibiotics and better medical care and

TABLE II.

The Death Rates in Australia from Acute Infective Disease.

Period.	Sex.	Death Rates per Million per Annum at Ages (Years) Given.								
		0 to 4	5 to 14	15 to 24	25 to 34	35 to 44	45 to 54	55 to 64	65 to 74	75 and Over
1908 to 1910 ..	M.	9284	570	505	581	558	693	1142	2705	6622
1911 to 1920 ..	M.	8564	609	602	976	932	958	1118	1978	5418
1921 to 1930 ..	M.	5134	334	219	211	302	416	642	1171	3572
1931 to 1940 ..	M.	2271	286	136	137	177	266	426	766	2088
1941 to 1945 ..	M.	1770	249	94	72	110	187	279	519	1309
1908 to 1910 ..	F.	7998	570	360	337	382	451	820	2446	6699
1911 to 1920 ..	F.	7275	635	430	616	570	630	954	2093	5813
1921 to 1930 ..	F.	4322	312	173	193	246	321	494	1106	3579
1931 to 1940 ..	F.	2056	249	90	116	149	200	310	675	2043
1941 to 1945 ..	F.	1541	183	73	80	95	127	186	412	1427

TABLE III.

The Death Rates in Australia from Acute Infective Disease in Childhood.

Period.	Sex.	Death Rates per Million per Annum at Ages (Years) Given.								
		0	1	2	3	4	1 to 4	5 to 9	10 to 14	All Ages.
1908 to 1910 ..	M.	28,686	8675	2530	1747	1255	3639	762	366	1759
1911 to 1920 ..	M.	23,128	8196	3050	2123	1517	3777	833	361	1785
1921 to 1930 ..	M.	14,355	5653	1970	1373	947	2505	457	204	893
1931 to 1940 ..	M.	5252	2657	1368	1058	727	1449	428	150	450
1941 to 1945 ..	M.	4085	1921	971	733	616	1082	354	148	342
1908 to 1910 ..	F.	25,430	7705	2698	1653	1172	3369	757	371	1510
1911 to 1920 ..	F.	19,322	7486	2863	1975	1601	3528	885	361	1482
1921 to 1930 ..	F.	11,873	5011	1831	1207	861	2245	440	180	768
1931 to 1940 ..	F.	4631	2549	1246	865	779	1356	373	132	392
1941 to 1945 ..	F.	3398	1910	877	641	475	997	260	109	293

improved hygiene, including public health measures. But these explanations are too limited and a broader ecological view of the problem should be taken. The antibiotics have come into general use in Australia only since 1945, and their effect on mortality before that year must be considered as negligible. Nor were the sulphonamide drugs in common use before 1940. It is probable that only minor changes in the treatment of the acute infective diseases took place over the years 1908 to 1945. Diphtheria, perhaps, is the only disease of the group for which effective specific treatment had been devised. We must, therefore, look to causes other than those affecting individual patients. General reviews of some of the factors involved in this almost world-wide decline in mortality have been given by Burnet (1946) and by Greenwood (1932, 1935 and 1936).

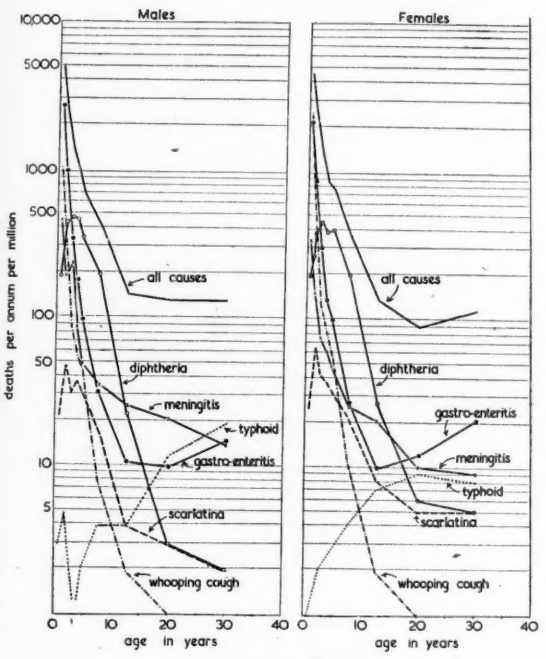

FIGURE I.

The mortality in Australia from certain of the acute infective diseases by age and sex for the years 1931 to 1940. ("All causes" is to be taken as "all acute infective diseases".)

One important factor in the incidence and mortality of the droplet-spread infections, such as diphtheria, pertussis and measles, is family size. This has been studied in some detail by the Harvard school (Aycock and Eaton, 1925; Wilson, Bennett, Allen and Worcester, 1939). Children in arms do not have much opportunity of acquiring disease from infants or children other than those in their own family. An important source of infection for an infant is, then, an elder member of the family.

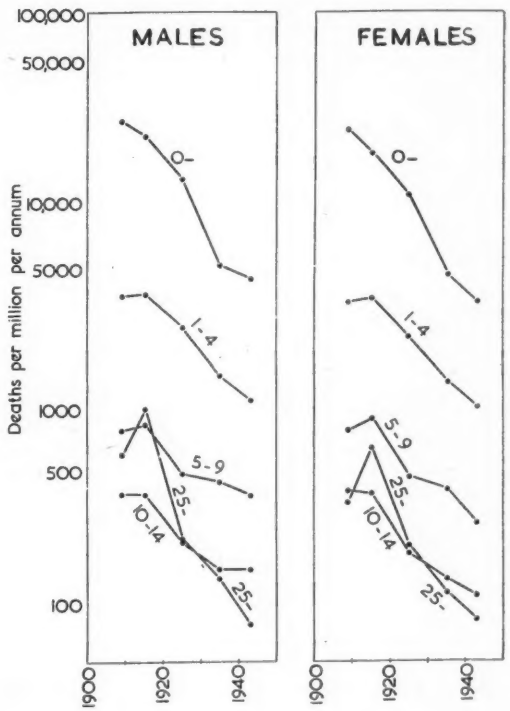

FIGURE II.

The trend of mortality in Australia from the acute infective diseases.

Adults are likely to be immune, but children of school age may not be. So a common finding is that the primarily infected subject in a household is a child of school age, and the next is an infant or younger sibling. Thus it is found that the age distribution of the secondary cases has a mean some years lower than that of the primary cases. Therefore, infants with older siblings face a hazard from the infective diseases greater than children of one-child families or the earlier members of a family. A change in family size may then affect the age at which children acquire the common droplet-spread diseases. There was a considerable decline in family size in Aus-

tralia over the years considered, and so this factor must be regarded as important.

Even in the gastro-intestinal infections factors other than public health activities have been of great importance. In Sydney, for example, the pasteurization of milk is not compulsory, although it is carried out by the larger distributors in order to be able to store the milk for some days before delivery. Milk-borne epidemics of diarrhœa and other diseases usually occur on the runs of the smaller distributors, who do not hold milk and hence do not find pasteurization necessary. Greenwood (1936) and Yule in the discussion on Greenwood's address have both emphasized the fact that many improvements in sanitation have arisen for hedonistic reasons and have added to the

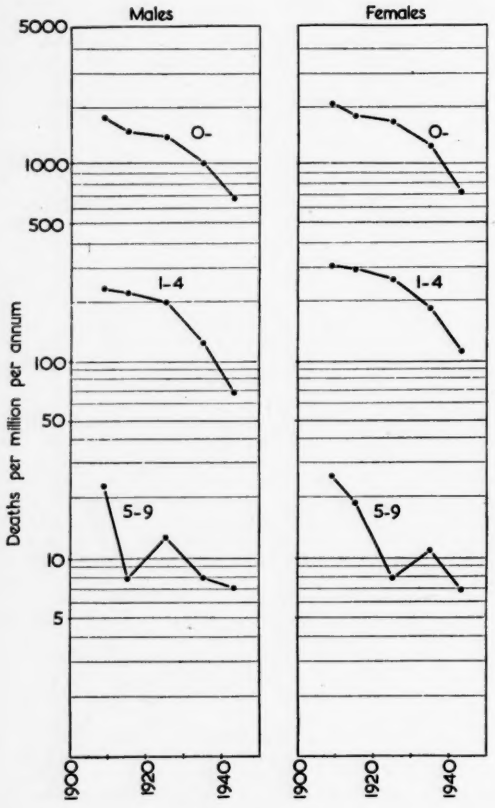

FIGURE III.
The trend of mortality in Australia from pertussis (whooping-cough).

amenities of life. One may cite the water-closet. Lice have become rare in the community without attention being directed to the possibility of the louse carrying typhus fever. The motor-car has ousted the horse, and so the fly has waned as a menace to health in the cities. This is held to be an important factor in the decline in incidence of the gastro-intestinal infections. Other plausible factors could be mentioned, such as dietary changes leading to increased resistance of populations of a non-specific nature against disease.

Consideration of Individual Diseases.

I shall now discuss the mortality from each of the diseases individually. Space will permit only pertussis to be discussed in full here. Consideration of the others must be deferred to a later paper.

The Mortality from Pertussis (Whooping-Cough) in Australia.

An historical account of pertussis in Australia from the time of the first European colonization has been given by Cumpston (1927). The death rates from pertussis are shown in Table VII. For any period, such as that from 1931 to 1940, the mortality is practically confined to the first few years of life. Thus, for males, the mortality

TABLE IV.
The Crude and Standardized Mortality Rates in Australia from the Group of Acute Infective Diseases.

Period.	Sex.	Deaths per Million per Annum from Acute Infective Diseases.			
		Crude Death Rate.	As Standardized onto the Enumerated Population of England and Wales, 1901.	As Standardized onto the Life Table Populations Derived from the Australian Census of 1933.	As an Equivalent Average Death Rate for Persons under the Age of 65 Years.
1908 to 1910	M.	1759	1765	1710	1337
1911 to 1920	M.	1785	1802	1706	1458
1921 to 1930	M.	893	943	899	722
1931 to 1940	M.	450	496	502	394
1941 to 1945	M.	342	370	357	289
1908 to 1910	F.	1510	1456	1543	1064
1911 to 1920	F.	1482	1470	1528	1150
1921 to 1930	F.	768	792	832	600
1931 to 1940	F.	392	426	465	330
1941 to 1945	F.	293	307	324	233

rate was 1009 per million for the first year of life, 312 for the second, 93 for the third, and 56 and 45 for the fourth and fifth years of life. After the age of five years mortality from pertussis is negligible. A similar conclusion may be reached after studying the female rates. Deaths from pertussis include the mortality from bronchopneumonia following the disease, if pertussis has been

TABLE V.
The Decline in the Crude and Standardized Death Rates from all Causes Compared with the Decline in the Mortality Rate from the Acute Infective Diseases, Given as the Difference between the 1908 to 1910 Rates and the 1941 to 1945 Rates.

Subjects.	Deaths per Thousand per Annum.			
	Crude Death Rates.	As Standardized onto the Population of England and Wales, 1901.	As Standardized onto the Life Table Population.	As Equivalent Average Death Rate.
Males:				
All causes	0·8	2·6	3·5	3·4
Infective diseases ..	1·4	1·4	1·4	1·0
Females:				
All causes	0·3	3·3	3·5	3·1
Infective diseases ..	1·2	1·1	1·2	0·7

mentioned on the death certificate. However, later deaths from bronchiectasis will not be included, even if pertussis has been mentioned, since the practice of the statisticians responsible for the design of the International List is rather to ignore very long-standing diseases, which have arisen either in infancy or congenitally.

Pertussis is remarkable, being one of the few infective diseases with a low masculinity for the death rates. This has already been noted in Table VI. The reasons for this low masculinity are not understood. The subject was discussed at a meeting of the Royal Statistical Society (Hill, 1933), at which it was shown that this low masculinity is a feature of the mortality in all countries under very variable climatic and hygienic conditions.

This low masculinity for pertussis is reflected in low masculinity rates for bronchiectasis, a not uncommon sequel, the importance of which has been stressed by Hirschfeld (1951). The official statistics are an unsatisfactory source of mortality data for bronchiectasis, for these deaths are referred to abscess of the lung, chronic bronchitis or tuberculosis of the lung. Nor will these deaths be referred to pertussis, as has already been pointed out. I have, therefore, obtained through the courtesy of Professor L. Dods the following information on admissions to two of the large Sydney hospitals for the years 1946 to 1949. At the Royal Alexandra Hospital for Children 164 children under the age of fourteen years were admitted with bronchiectasis (multiple admissions of the same child were counted only once), and of these 57 were males and 107 females. Similarly, at the Royal Prince Alfred Hospital, with patients over fifteen years of age 336 patients were admitted, of whom 130 were males and 206 females. If we suppose that the populations at risk for the two sexes were approximately equal for either hospital, the masculinity of the admissions may be computed. At the Royal Alexandra Hospital for Children the masculinity was 0·53, and at the Royal Prince Alfred Hospital it was 0·63. This finding of a low masculinity rate of bronchiectasis suggests that numerically pertussis may be the predominant cause of bronchiectasis in the population served by these two hospitals, unless some other diseases with low masculinity are also involved.

If we compare the mortality rates for given age groups and either sex over the years of the survey, 1908 to 1945, a great improvement is apparent. Such comparisons may be made either in Table VII or in Figure III. The causes for this decline are by no means certain. There has been no efficient prophylactic immunization, nor can changes in therapy have had much effect, since the decline appeared before 1931. It is possible that the most important factors were the general improvement in child welfare and the coming of the small family, as pointed out in an earlier section of this paper.

TABLE VI.

The Masculinity[1] of the Death Rates from Certain Diseases in Australia in the Years 1931 to 1940.

Disease.	The Masculinity[1] of the Death Rate at Ages (Years) Given.			
	0	1 to 4	5 to 9	10 to 14
Meningitis and encephalitis	138	135	138	124
Pertussis	81	67	—[2]	—[2]
Diphtheria	106	108	100	85
Measles	93	105	—[2]	—[2]
Influenza	118	196	—[2]	—[2]
Gastro-enteritis	130	105	—[2]	—[2]
All diseases of the group..	113	107	115	114

[1] The masculinity has been defined as 100 times the male death rate divided by the corresponding female rate.

[2] The rates were too low in these cases to make the masculinity reliable.

More information should be prepared in Australia on this important disease. However, it is notifiable only in South Australia, the Northern Territory and the Australian Capital Territory, so that figures on the incidence of the disease are available for but a small proportion of the total Australian population. This position may be contrasted with the custom in England and Wales of preparing "corrected notification" statistics and relating them to the death statistics. A table from a recent report of the Registrar-General (1949) is reproduced here as Table VIII, in order to draw attention to the need for similar data for Australia. The incidence of the disease as measured by the notifications is greatest at ages three and four years last birthday, somewhat less at ages one and two years, and still less in the first year of life. In the last group the incidence is about two-thirds of the incidence at ages three and four years. The case fatality rate is highest in the first year of life and decline with increasing age. Thus in 1944 the case fatality rate was 62·6 per thousand for males in the first year of life and 72·1 for females. At ages one and two last birthday the case fatality rates were 9·4 and 13·0 per thousand, at ages three and four years 0·9 and 3·0 per thousand for males and females respectively. At ages five to nine years the rates were 0·9 and 1·1 per thousand for the two sexes. These figures show, firstly, that there is not only an increased incidence of the disease among females, but also that the case fatality rate is higher in this sex. Secondly, the case fatality rate declines very steeply with age, so that any factor which causes a delay in the acquisition of the disease by children will result in a lowered mortality rate. Thus it is apparent from the English figures that even postponing infections to the second year of life might be expected to reduce very considerably the number of deaths due to pertussis. Thirdly, the figures show that preventive measures must be directed at children in the

TABLE VIII.

The Case Fatality Rates for Whooping-Cough in England and Wales by Age and Sex, 1944 and 1945. (From the Registrar-General's Statistical Review of England and Wales for the Six Years 1940–1945, Volume I, Table XL.V)

Ages. (Years.)	Notification per 1,000 Living.				Deaths per 1,000 Notifications.			
	1944.		1945.		1944.		1945.	
	M.	F.	M.	F.	M.	F.	M.	F.
0—	14·5	15·1	9·4	10·0	62·6	72·1	58·2	64·9
1—	19·4	22·0	12·7	14·4	9·4	13·0	9·1	14·1
3—	20·7	24·1	14·2	16·5	0·9	3·0	1·7	3·4
5—	9·8	11·2	6·0	7·0	0·9	1·1	0·7	1·5
10—	0·8	1·0	0·5	0·6	0·9	—[1]	1·4	—
15 and over ..	0·0[1]	0·1	0·0	0·0	2·7	1·6	—	1·3
All ages ..	2·7	2·3	1·8	1·5	10·3	12·0	9·9	12·0

[1] "0" means less than 0·05; "—" means no death occurred in the particular instance.

TABLE VII.

The Mortality from Whooping-Cough (Pertussis) in Australia.

Period.	Sex.	Deaths from Pertussis per Million per Annum at Ages (Years).								
		0	1	2	3	4	1 to 4	0 to 4	5 to 9	10 to 14
1908 to 1910 ..	M.	1735	640	147	55	71	235	573	24	2
1911 to 1920 ..	M.	1456	550	186	84	48	221	526	8	1
1921 to 1930 ..	M.	1358	525	135	66	37	193	450	13	2
1931 to 1940 ..	M.	1009	312	93	56	45	126	313	8	2
1941 to 1945 ..	M.	672	179	53	20	13	69	208	7	1
1908 to 1910 ..	F.	2020	717	247	109	116	303	662	26	3
1911 to 1920 ..	F.	1751	708	265	121	45	290	635	19	1
1921 to 1930 ..	F.	1672	647	229	93	45	256	561	8	—
1931 to 1940 ..	F.	1250	483	124	85	57	187	410	11	2
1941 to 1945 ..	F.	729	270	91	51	17	112	252	7	1

first year of life. Collection of such data as those of Table VIII should not be regarded as research, but as a necessary function of the public health departments, for without them no conclusions can be made as to the efficacy of immunization campaigns and specific therapies.

In conclusion, let me quote, after Bradford Hill (1933), from the Fourth Epidemiological Report of the League of Nations (1926):

> The opinion, often met with and reflected in the regulations for the notifications of these diseases, that whooping-cough and measles are less important than scarlet fever and diphtheria, evidently dates from the latter half of the nineteenth century, when the two latter diseases caused many more deaths than they do now, and when the mortality under two years of age was so heavy as to leave less in evidence such causes as measles and whooping-cough.

Conclusion.

The discussion of the other diseases of the group of infective diseases must be postponed to a later paper. A summary of conclusions for the whole group of acute infective diseases will be given there.

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OBSERVATIONS ON THE INCIDENCE AND COMPOSITION OF URINARY CALCULI.

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THE occurrence of urinary calculi in man has been recognized for thousands of years. It was so well known at the time of Hippocrates that in his famous oath he insists that the surgical treatment of the condition be restricted to specialists. The first known example of a urinary calculus seems to be that found by Elliott Smith in a predynastic tomb in upper Egypt. The date of the tomb has been estimated by Flinders Petrie to be at least 5000 B.C. This calculus was examined by Shattock (1905). It has an inner portion composed of ammonium urate surrounded by an outer zone composed of phosphates of calcium, magnesium and ammonium. In these and other respects it is indistinguishable from similar types of calculus which are found today.

The first detailed chemical analyses of urinary calculi were carried out by Wollaston (1796) over a hundred and fifty years ago. He recognized the occurrence of oxalate, phosphate and urate calculi, and showed that calcium, magnesium and ammonium might be among the bases present. In 1810 he added cystine calculi to the list, and revised his classification to the form which is in use at the present day.

In 1798 Pearson published analyses and descriptions of some 200 calculi. He first introduced the name uric acid for the substance which had been named lithic acid by its discoverer Scheele a few years earlier.

Many urinary calculi have been described in the intervening period, but only a small proportion of them were submitted to chemical analysis. Although surgeons were reluctant to submit their trophies to the destructive attentions of the chemist, they frequently did not hesitate to draw conclusions concerning the composition of the stones. These conclusions, we now have good reason to believe, were often incorrect. Attention was directed to this state of affairs by Benjamin Moore, the first incumbent of a British chair of biochemistry. Mackerell, Moore and Thomas (1911) published the analyses of 24 urinary calculi and showed that current ideas, based on appearance and physical properties, were mistaken as to the relative plentifulness of calculi of different composition. Oxalates, not urates, were found to be most plentiful. Urates were found to be the least plentiful of the three main classes of calculi. This conclusion was based on a small number of analyses, but it has since been confirmed by the results of hundreds of analyses from many sources. The previous erroneous ideas may have been derived from mistaken interpretation of Wollaston's work. He recognized that many calculi were of mixed composition, and claimed to have found traces of uric acid in most of them.

The classification of urinary calculi introduced by Wollaston, and still in use, is based on the nature of the anions occurring in them. However, McIntosh (1942) contends that, apart from certain organic calculi (uric acid, cystine), the disturbance resulting in urinary calculosis is primarily of calcium metabolism. Magnesium and ammonium may become involved secondarily, but their occurrence and the nature of the anion are essentially a function of the reaction of the urine. The contentions of McIntosh are well founded, but the current classification is probably still the more convenient for practical purposes.

The ætiology of urinary calculosis has been the subject of a great deal of investigation. Extensive studies have been made of the more obvious factors which may affect it. Age, sex, race and diet have been considered. Environmental factors—climatic, seasonal, geographical and geological—have been examined. The studies up to 1929 have been reviewed by Joly, who shows that the findings have never been clear-cut, but have often been contradictory. On one point only is there general agreement: that in western Europe and in America the age incidence of urinary calculosis has changed within the last century or less. From being mainly a disease of childhood, it has become mainly a disease of middle age. This change does not seem to have occurred in eastern European or Asiatic countries.

In the present study of the incidence of urinary calculosis in the population of a general hospital, two groups have been examined. The first group comprised 27,480 males and 25,680 females admitted to Sydney Hospital over a period of about eight years, from 1942 to 1950. It does not include patients admitted to the eye department of the hospital. It contains a much smaller proportion of children than the population at large. In this group over 400 cases of urinary calculosis occur. They have been examined for distribution with respect to age, sex and site of occurrence of the calculus in the urinary tract.

TABLE I.

Age and Sex Incidence of Urinary Calculi.

Age. (Years.)	Males.				Females.			
	Admissions.		Calculi.		Admissions.		Calculi.	
	Number.	Percentage of Total.	Number.	Percentage of Decade.	Number.	Percentage of Total.	Number.	Percentage of Decade.
10	1952	7·10	9	0	1194	6·70	0	0
20	2419	8·80	11	0·455	2466	9·60	5	0·203
30	3518	12·80	31	0·882	4275	16·64	39	0·912
40	3243	11·80	54	1·666	4366	17·00	35	0·802
50	3876	14·10	80	2·064	4289	16·70	30	0·709
60	4892	17·80	53	1·084	4084	15·90	11	0·269
70	4810	17·50	39	0·811	3070	12·30	10	0·326
80	2254	8·20	11	0·488	1078	4·20	2	0·186
90	440	1·67	1	0·228	257	1·00	0	0

From the second group over 300 calculi were obtained for analysis over a period of some twenty-five years. This group has been submitted to an examination similar to the first, with the addition of an examination of the distribution of chemical composition. The proportions in the total hospital population have not been calculated, as the figures for this period are not available.

The groups of analysed and diagnosed calculi are not mutually exclusive. The data for analysed calculi were collected over a much longer period than those for diagnosed calculi. About half of the calculi observed by diagnosis were obtained for analysis. About one-third of the analysed calculi were examined in the period before that for which the data for diagnosed calculi are available.

The analysed group is the result of a different method of selection from the diagnosed group. Table III and Figure II show that while the general distribution of the two groups in the urinary tract is similar, a larger proportion of calculi were obtained from the lower portions of the urinary tract for analysis. This may be due to the accessibility of calculi from lower parts of the urinary tract. A number of these calculi were passed spontaneously, others were obtained by relatively minor procedures. Access to calculi in the upper portions of the tract, on the other hand, can be obtained by major surgical operations. Another factor in the selection of calculi for analysis may be a tendency on the part of the surgeon to pick out "interesting" calculi for analysis.

Small discrepancies in the totals of analysed calculi considered in different categories (for example, with respect to age and site of occurrence, or to age and composition) are due to the fact that complete information as to site and age was not available for some of the calculi examined.

Sex and Age Distribution of Urinary Calculi in a General Hospital.

The numbers of admissions by decades of age are shown in the second column of Table I, the percentages of the total admissions in the third column of the table. The numbers of calculi diagnosed in each decade are shown in the fourth column, the percentages of the total number of calculi in the fifth column. The percentages of admissions in each decade in which urinary calculi were diagnosed are shown in the sixth column.

The figures in Table I show that 280 urinary calculi were diagnosed in 27,480 males, or 1·019%, and 132 in 25,680 females, or 0·514%. However, the percentages of urinary calculi diagnosed vary considerably with age. As the number of admissions to hospital varies with age also, the manner of variation is not the same in the two sexes. Among males the admissions to hospital rise slowly to their maximum in the sixth and seventh decades and then decline sharply. Among females the admissions to hospital rise to a figure in the third decade from which they vary little for four decades, and then fall.

The percentages of urinary calculi diagnosed in males rise from nothing in the first decade to 2·06 in the fifth decade. This maximum is about twice the general rate of occurrence of calculi. It falls to 0·23% in the ninth decade.

In females the percentages of calculi diagnosed rise from nothing in the first decade to 0·91 in the third decade; from this there is a slight decline for the next two decades, and then a more rapid fall followed by a slight rise in the seventh decade, to nothing in the ninth decade. At no time does the percentage occurrence exceed the general rate of occurrence as it does in males.

The figures in the fifth and ninth columns of Table I have been plotted against age in Figure I.

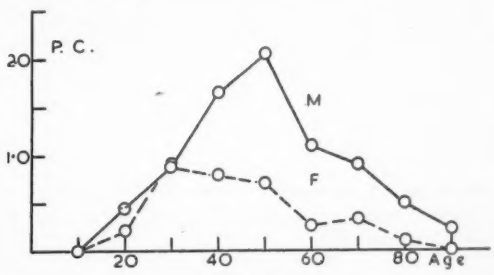

FIGURE I.

Distribution with age of urinary calculi in 53,160 consecutive admissions to Sydney Hospital (27,480 male subjects, 25,680 female subjects). Abscissæ: age by decades; ordinates: percentages of total numbers.

While for the whole group urinary calculi are twice as frequent in males as in females, this ratio varied considerably from decade to decade. It is approximately 1:1 (0·97) in the third decade, while it is approximately 3:1 (2·95) in the fifth decade. In the other decades it ranges from 2·07 to 2·63.

The age incidence of urinary calculi may also be considered from the proportion of total calculi occurring in each decade. This has been done in Table II, both for the first series and for the second series (calculi analysed).

The information given in Table II is essentially that shown in the fourth, fifth, eighth and ninth columns of Table I, but it makes possible a comparison between the first series (diagnosed) and the second series (analysed) of calculi. The age distribution does not differ materially between the two series over most of the range. In both series of males most of the calculi occur in the fourth, fifth and sixth decades (66·85% and 59·66% respectively), although the peak in the fifth decade does not occur in the second series. In both series of females most (78·8% and 65·92% respectively) occur in the third, fourth and fifth decades, but as with the males, the proportion in the three decades is not so great in the second as in the first series. A striking difference occurs in the ninth decade. No calculi occur in this in the first somewhat larger series, but in the second series 11 calculi, or 12·5% of the total, are found in this decade.

The difference between the proportions of males to females in the two series is not significant. The differences of distribution among the successive decades in the two series also are not significant for the males, nor for the females except for the ninth decade.

TABLE II

Age and Sex Distribution of Urinary Calculi.

Age. (Years.)	Males. Number.	Males. Percentage of Total.	Females. Number.	Females. Percentage of Total.
Calculi Diagnosed.				
10	0	0	0	0
20	11	3·93	5	3·79
30	31	11·07	39	29·55
40	54	19·29	35	26·52
50	80	28·58	30	22·73
60	53	18·98	11	8·34
70	39	13·93	10	7·58
80	11	3·93	2	1·52
90	1	0·36	0	0
Total	280		132	
Calculi Analysed.				
10	3	1·32	1	1·14
20	3	1·32	0	0
30	29	12·72	22	25·00
40	43	18·86	16	18·19
50	52	22·81	20	22·73
60	46	20·17	11	12·50
70	30	13·16	5	5·68
80	19	8·33	2	2·27
90	3	1·32	11	12·50
Total	228		88	

Distribution of Calculi in the Urinary Tract.

The calculi have been grouped according to whether they were found in the kidney, the ureter, the bladder, or the urethra or passed in the urine. For certain purposes examination in two groups, calculi from the upper and those from the lower part of the urinary tract, gives more useful information. The numerical and percentage distributions in the various groups are shown separately for diagnosed and for analysed calculi, and for the two sexes, in Table III.

Table III shows that by far the largest proportion of calculi found by diagnosis occurs in the upper part of the urinary tract—nearly 80% in males and over 90% in females. Of each of these groups renal calculi form about two-thirds. Of the calculi diagnosed in the lower part of the urinary tract, over 90% occurred in the bladder in males, and all of them occurred in the bladder in females. The difference of distribution in the upper and lower parts of the urinary tracts between the two sexes is statistically significant at the $P = 0{\cdot}005$ level at least.

Most of the calculi obtained for analysis also were found in the upper part of the urinary tract—nearly 60% in males and nearly 75% in females. The proportion of ureteric to renal calculi is higher among the males but lower among the females than in the diagnosed calculi. The significance of this difference of distribution between the two sexes lies between the $P = 0{\cdot}005$ and $P = 0{\cdot}05$ levels for this group. The distribution of analysed calculi between the upper and lower parts of the urinary tract does not differ significantly from the 50% distribution of chance in males; in females the difference is significant at better than the $P = 0{\cdot}05$ level.

The higher proportion of calculi from the lower part of the urinary tract among calculi obtained for analysis is due mainly to the much higher proportion of urethral or passed calculi in this group. The higher proportion of vesical calculi in males than in females in both groups may be associated with the greater difficulty of passage of calculi from the bladder in males.

The higher proportion of urethral and passed calculi obtained for analysis in both sexes, as compared with those observed indirectly, may be due to the fact that the occurrence and passage of urethral calculi are often accompanied by pain. A larger proportion of patients with this condition would be likely to seek medical advice, and from them calculi are readily obtained for analysis. Calculi obtained for analysis and those located in diagnosis are therefore derived from differently selected groups of patients.

TABLE III.

Topographical Distribution of Urinary Calculi.

Site.	Males. Number.	Males. Percentage of Total.	Females. Number.	Females. Percentage of Total.
Sydney Hospital Analysed Group, 1925 to 1950:				
Upper[1]:				
Renal ..	82	35·35 } 60·35	47	53·4 } 67·05
Ureteric ..	58	25·00 }	12	13·65 }
Lower[1]:				
Vesical ..	54	23·3 } 39·65	17	19·3 } 32·95
Urethral ..	38	16·35 }	12	13·65 }
Sydney Hospital Diagnosed Group, 1942 to 1950:				
Upper:				
Renal ..	145	51·8 } 79·3	90	68·2 } 92·45
Ureteric ..	77	27·5 }	32	24·25 }
Lower:				
Vesical ..	53	18·95 } 20·75	10	7·6 } 7·6
Urethral ..	5	1·8 }	0	0 }
London Hospital Group (Lett), 1905 to 1934:				
Upper:				
Renal ..	849	46·3 } 65·6	719	75·85 } 94·75
Ureteric ..	353	19·3 }	179	18·9 }
Lower:				
Vesical ..	558	30·4 } 34·4	50	5·3 } 5·3
Urethral ..	73	4·0 }	0	0 }
St. Peter's Hospital Group (Lett), 1915 to 1924:				
Upper:				
Renal ..	165	27·4 } 32·7		
Ureteric ..	32	5·3 }		
Lower:				
Vesical ..	371	61·5 } 67·1		
Urethral ..	34	5·6 }		
U.S.A., Several Hospitals (Domanski), 1940:				
Upper:				
Renal ..	44	30·1 } 65·3	49	65·3 } 96·3
Ureteric ..	52	35·2 }	23	31·0 }
Lower:				
Vesical ..	51	34·7 } 34·7	2	2·7 } 2·7
Urethral ..	—	— }	—	— }

[1] "Upper", upper part of urinary tract; "lower", lower part of urinary tract.

Similar distributions along the urinary tract were found by Lett (1936), by Winsbury-White (1938), by Domanski (1940), and by McKay, Baird and Lynch (1948). Lett and Winsbury-White also found the incidence of urinary calculi to be about twice as great in males as in females. Joly (1929), in a series of 602 calculi, found about twice as many in the bladder and urethra as in the upper parts of the urinary tract. This difference is attributed by Lett to the fact that Joly's series was observed in a special hospital (Saint Peter's Hospital for the Stone), to which a selected group of patients would go, and is made up predominantly of males, in whom calculi are commoner in the lower part of the urinary tract. He points out that symptoms arising from vesical calculus are usually correctly localized by the patient, while he cannot so readily localize symptoms arising from renal calculi. The other three series, like the present series, were derived from general hospitals.

TABLE IV.

Age Distribution of Calculi from Different Parts of Urinary Tract.

Age. (Years.)	Renal.		Ureteric.		Vesical.		Urethral and Passed.	
	Number.	Percentage of Total.	Number.	Percentage of Total.	Number.	Percentage of Total.	Number.	Percentage of Total.
Calculi from Males Analysed.								
10	0	0	0	0	0	0	3	1·35
20	2	0·9	2	0·90	1	0·45	0	0
30	9	4·05	10	4·59	1	0·45	0	0
40	25	11·2	8	3·6	1	0·45	10	4·6
50	19	8·5	18	8·05	6	2·7	5	2·25
60	15	6·7	13	5·85	13	5·85	6	2·7
70	4	1·8	4	1·8	18	8·05	8	3·6
80	1	0·45	0	0	16	7·2	2	0·9
90	1	0·45	0	0	2	0·9	0	0
Sum ..		34·05		24·8		26·05		15·4
Calculi from Females Analysed.								
10	1	1·3	0	0	0	0	0	0
20	0	0	0	0	0	0	0	0
30	11	14·1	6	7·7	1	1·3	2	2·55
40	6	7·7	2	2·55	1	1·3	4	5·1
50	11	14·1	2	2·55	1	1·3	3	3·85
60	9	11·55	0	0	2	2·55	0	0
70	3	3·85	0	0	0	0	2	2·55
80	1	1·3	1	1·3	0	0	0	0
90	5	6·4	1	1·3	2	2·55	2	2·55
Sum ..		60·25		15·4		9·0		15·4
Calculi Diagnosed in Males.								
10	0	0	0	0	0	0	0	0
20	6	2·2	5	1·85	0	0	0	0
30	16	5·9	14	5·2	0	0	1	0·35
40	38	14·05	10	3·7	5	1·85	1	0·35
50	35	12·95	28	10·35	6	2·2	1	0·35
60	23	8·5	12	4·45	17	6·3	1	0·35
70	14	5·2	8	2·95	16	5·9	1	0·35
80	3	1·1	0	0	8	2·95	0	0
90	0	0	0	0	1	0·35	0	0
Sum ..		50·0		28·5		19·55		1·85
Calculi Diagnosed in Females.								
10	0	0	0	0	0	0	0	0
20	5	3·8	0	0	0	0	0	0
30	22	16·8	15	11·45	2	1·5	0	0
40	23	17·55	9	6·85	3	2·3	0	0
50	22	16·8	5	3·8	3	2·3	0	0
60	9	6·85	1	0·75	1	0·75	0	0
70	7	5·35	2	1·5	0	0	0	0
80	2	1·5	0	0	0	0	0	0
90	0	0	0	0	0	0	0	0
Sum ..		68·7		24·4		6·85		0

The distributions of calculi in the London Hospital series and in the Saint Peter's Hospital series are shown in Table III for comparison.

Age and Site of Occurrence of Calculi.

The age distribution by decades of calculi occurring in different parts of the urinary tract is shown in Table IV.

The distributions of the upper and lower tract calculi with age are plotted graphically in Figure II. The values for females show a similar distribution in each decade to that in the whole groups of both diagnosed and analysed calculi. Most of the calculi of each group occur in the two decades between thirty and fifty years (81% and 71% of the upper urinary tract calculi, 68% and 89% of the lower tract calculi respectively).

Among males the distribution of calculi from the upper part of the urinary tract with age differs from that of calculi from the lower part of the urinary tract. The difference is similar for diagnosed and analysed calculi. Most of the upper urinary tract calculi occur in the third, fourth and fifth decades (69% and 75%). Most of the lower tract calculi occur in the sixth, seventh and eighth decades for diagnosed, and in the seventh, eighth and ninth decades for analysed calculi (91·5% and 68%).

If the calculi are divided into those occurring before and after the age of fifty years respectively, the different distribution of upper and lower renal calculi is found to be highly significant statistically ($P < 0.005$). Calculi diagnosed among females have the following distribution between these two age groups: renal calculi are slightly less frequent in the older group than in the younger group, ureteric calculi are about a third as frequent in the older group, the very small number of vesical calculi is equally distributed; no urethral or passed calculi are recorded.

Distribution of Composition with Age and Sex.

Although the classification of urinary calculi according to the nature of the anion may depend primarily upon the reaction of the urine rather than upon the underlying metabolic disturbance, this classification remains convenient for general use. Only the three major groups, oxalate, phosphate and urate (including uric acid) calculi, are considered in Table V. The rarer calculi, such as cystine calculi, of which only four occur in the whole series, are

not considered in the discussion of distribution. Further, it has been preferred to limit the survey to those calculi which may be considered to be derived from common normal constituents of the urine as a result of abnormal changes of solubility or concentration, and to exclude those which can be regarded as the result of abnormal prerenal metabolism.

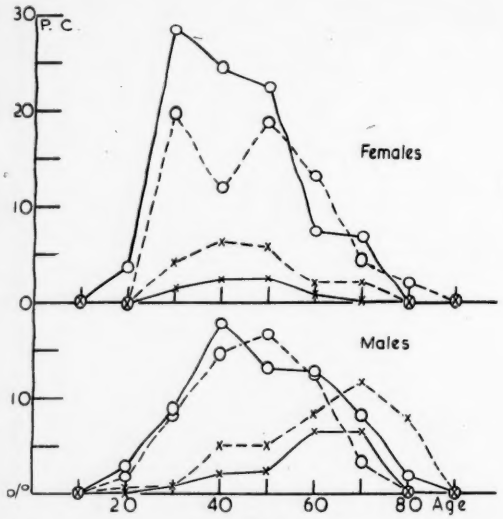

FIGURE II.

Distribution with age of urinary calculi in upper (kidney and ureters) and lower (bladder, urethra and "passed") parts of urinary tract. Abscissæ: age by decades; ordinates: percentage of total number. Continuous line, diagnosed calculi; interrupted line, analysed calculi; lines with crosses, calculi in lower part of urinary tract.

From Table V it can be seen that among males oxalate and phosphate calculi make up nearly 90% of the total. Oxalate calculi are somewhat, but not significantly, more numerous than phosphate calculi. The same general distribution holds for females. Oxalate and phosphate calculi form over 90% of the total. The numbers of each are almost the same. For both sexes and for the whole range of age, urate calculi form only small proportions of the totals.

The numbers of phosphate and of oxalate calculi in each sex vary in the same general manner as the calculi as a whole without respect to composition. The maxima for oxalate tend to occur somewhat earlier in oxalate than in phosphate calculi, in the fourth and fifth rather than in the fifth and sixth decades. For urate calculi the maxima occur in the fifth decade. However, in women the number increases again in extreme old age. The numbers are too small to allow definite conclusions to be drawn from them.

The variation of composition with age is shown graphically in Figure III.

Mixed Calculi.

Many urinary calculi are of mixed composition. They often consist of an inner portion which forms the primary calculus, surrounded by a layer of varying thickness of different composition, or there may be alternating layers differing in composition. These changes are usually the result of changes in the reaction of the urine. Among the factors which may cause changes of reaction are infection of the urinary tract, alteration of acid-base balance resulting from change of diet, and therapy.

In the present examination mixed calculi have been classified according to the composition of the primary portion, when this forms the bulk of the calculus. When the nucleus forms only a small proportion of the whole, the classification has been according to the composition of the major part.

When phosphates are deposited on calculi which consist initially of calcium oxalate or of urate (including uric acid), there has been a rise in the *p*H of the urine during the period of deposition of the phosphate. When magnesium appears in phosphate calculi, it is an indication of increase of concentration of ammonia in already alkaline urine. It has been suggested that such changes of composition are associated with the occurrence of secondary infection in the urinary tract. In examinations of the composition of urinary calculi over a long period, it was noticed that the occurrence of these mixed calculi became less frequent from 1940, when sulphonamide therapy was introduced.

TABLE V.

Age Distributions of Calculi of Different Compositions.

Age. (Yrs.)	Oxalate. Number.	Oxalate. Percentage of Total.	Phosphate. Number.	Phosphate. Percentage of Total.	Urate. Number.	Urate. Percentage of Total.
			Males.			
10	0	0	3	1·15	0	0
20	5	1·9	1	0·4	0	0
30	27	10·25	6	2·4	4	1·5
40	35	13·3	16	6·1	5	1·9
50	28	10·65	24	9·1	7	2·65
60	22	8·35	23	8·75	5	1·9
70	15	5·7	9	3·4	6	2·3
80	9	3·4	6	2·3	4	1·5
90	1	0·4	1	0·4	1	0·4
Sum		53·95		33·9		12·15
			Females.			
10	1	1·1	0	0	0	0
20	0	0	7	7·8	0	0
30	12	13·2	7	7·8	0	0
40	10	11·1	7	7·8	1	1·1
50	8	8·9	9	10·0	3	3·3
60	6	6·7	5	5·6	1	1·1
70	2	2·2	3	3·3	0	0
80	1	1·1	3	3·3	1	1·1
90	3	3·3	0	0	0	0
Sum		47·8		45·55		6·65

In Table VI are shown the proportions of oxalate and urate calculi which contained phosphate, and of phosphate calculi which contained magnesium up to and after 1940.

TABLE VI.

Occurrence of Phosphate in Oxalate and in Urate Calculi, and of Magnesium in Phosphate Calculi, Before and After Introduction of Sulphonamide Therapy (Before and Since 1940).

Type of Calculi.	Before 1940.	Since 1940.
Oxalate and urate calculi:		
Phosphate present in	32 (39·1%)	20 (15·1%)
Phosphate not detected ..	50 (60·9%)	112 (84·9%)
Phosphate calculi:		
Magnesium present in	20 (66·7%)	42 (45·6%)
Magnesium not detected ..	10 (33·3%)	50 (54·4%)

The figures in Table VI show that the occurrence of phosphate in oxalate and urate calculi was more than twice as frequent before 1940 as after. The difference between the two periods is highly significant statistically ($P < 0.005$).

The figures also show that the occurrence of magnesium in phosphate calculi was over one and a half times as frequent before 1940 as it was after. This difference also is statistically significant, although not so highly significant as the former ($P < 0.05$). It is possible that these changes may be associated with a reduction of secondary infection as a result of sulphonamide therapy.

TABLE VII.

Site and Composition of Urinary Calculi.

Site of Calculi.	Oxalate.		Phosphate.		Urate.	
	Number.	Percentage of Total.	Number.	Percentage of Total.	Number.	Percentage of Total.
			Males.			
Upper[1]:						
Renal	55	22·65 } 40·35	19	7·8 } 13·15	6	2·5 } 3·75
Ureteric	43	17·7	13	5·35	3	1·25
Lower[1]:						
Vesical	26	10·7 } 18·5	24	9·9 } 16·9	13	5·35 } 7·4
Urethral	19	7·8	17	7·0	5	2·05
			Females.			
Upper:						
Renal	23	29·1 } 36·7	21	26·6 } 32·9	2	2·5 } 2·5
Ureteric	6	7·6	5	6·3	0	0
Lower:						
Vesical	3	3·8 } 12·65	3	3·8 } 8·85	2	2·5 } 6·3
Urethral	7	8·85	4	5·05	3	3·8

[1] "Upper", upper part of urinary tract; "lower", lower part of urinary tract.

Distribution of Composition with Site.

The distribution of calculi of different composition among different sites of occurrence is set out in Table VII. The figures in Table VII show that the proportions of calculi of different composition differ according to the site of occurrence. The proportions are expressed as percentages of total number of calculi. Among males nearly three-quarters of the renal and ureteric calculi were mainly calcium oxalate. Phosphate calculi were about one-third as plentiful, while urate calculi formed less than one-tenth of the total.

In vesical and urethral or passed calculi oxalates and phosphates were about equally prevalent and together formed about four-fifths of the total, while urates formed the rest and were about twice as prevalent as in the upper sites.

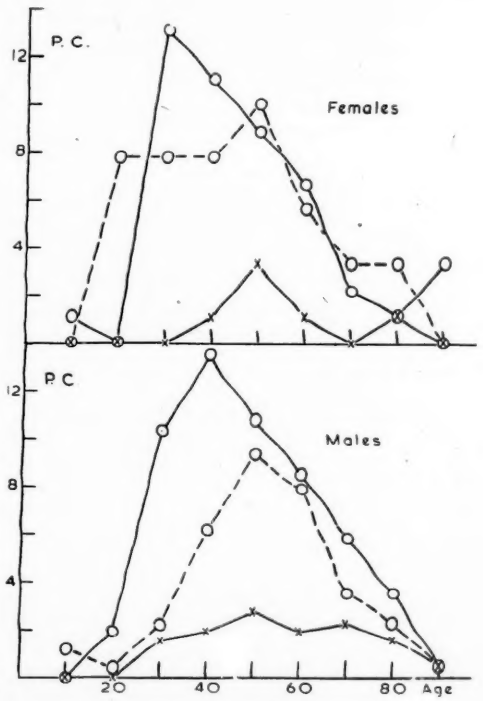

FIGURE III.

Distribution with age of composition of urinary calculi. Abscissæ: age by decades: ordinates: percentage of total number. Continuous line, oxalate; interrupted line, phosphate; continuous line with crosses, uric acid and urate.

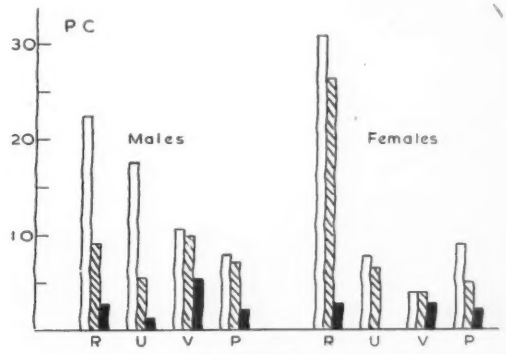

FIGURE IV.

Distribution of composition of urinary calculi according to position in urinary tract. Abscissæ: position in the urinary tract: *R*, renal; *U*, ureteric; *V*, vesical; *P*, urethral or passed. Open columns, oxalates; hatched columns, phosphates; black columns, urates and uric acid.

Among females oxalate and phosphates were about equally prevalent in renal, ureteric and vesical calculi. In renal and ureteric calculi hardly any urates occurred, while in vesical calculi they formed about one-fourth of the total.

In urethral and passed calculi oxalates were about twice as plentiful as phosphates. Urates formed again about one-fifth of the total. These distributions are shown graphically in Figure IV.

If attention is directed to the distribution of oxalate and phosphate calculi—that is, those resulting essentially from disturbances of calcium metabolism, and of quite different metabolic origin from urate calculi—Table VI is seen to contain some interesting information. For the purpose of this examination sites of occurrence have been grouped as upper (kidney and ureter) and lower (bladder and urethra) portions of the urinary tract.

Among males 98 oxalate calculi were from the upper part of the urinary tract and 45 from the lower part. The

corresponding numbers of phosphate calculi are 32 and 41. These distributions are significantly different statistically, the odds that the difference is not due to chance being over 200 to one ($P < 0.005$).

A similar examination of the calculi obtained from females shows a very different distribution. The numbers of oxalate calculi from the upper and lower parts of the urinary tract are 29 and 10 respectively, and of phosphate calculi, 26 and 7. The distribution of oxalates is obviously not significantly different from that of phosphates.

When the distributions of oxalate calculi between the upper and lower parts of the urinary tract of males and of females are compared, the difference is found not to be statistically significant. The distribution of phosphate calculi, on the other hand, is such that these are significantly more plentiful in the lower part of the urinary tract of males than in that site in females. The distribution of oxalate calculi is therefore significantly different from that of phosphate calculi in males, but not in females.

The table of Loewenthal and Wilson (1939) was used for obtaining the degree of significance of results.

Summary.

The incidence of urinary calculosis in the population of a general hospital has been examined. Of 27,480 males, 1·0%, and of 25,680 females, 0·5%, were affected.

The relation between age, sex, site of occurrence and composition have been examined. The maximum incidence tended to fall during the decades of middle age. Calculi were more frequent in the upper than in the lower parts of the urinary tract. Oxalates and phosphates made up about nine-tenths of the calculi examined, oxalates being the more plentiful. Urates and uric acid made up nearly all the rest.

The composition of mixed calculi showed a significant diminution of phosphate and of magnesium in the period following the introduction of sulphonamide therapy.

Acknowledgement.

The writer wishes to express his indebtedness to Dr. Ian D. Thomas, Out-Patient Registrar of Sydney Hospital, for much help in the extraction of information from the hospital records.

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TUBERCULOSIS AND THE GENERAL PRACTITIONER.[1]

By PHILIP WOODRUFF,

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MY subject is one of considerable importance to the community, and I think to the members of this Association. By common consent a number of branches of medical practice, both in this country and in other places with which we are familiar, have become in part the everyday functions of government health departments. The whole subject of tuberculosis control is, of course, one of these branches.

There is ample room for differences of opinion concerning the extent to which such a limited medical field should be, or should become, the responsibility of the State. But it is as well for us all to realize that any such specialty, however restricted and however much under government control it may be, must, in a community such as this, depend for its very existence on the broad field of private practice. I need hardly add that it is particularly necessary for those holding positions such as mine to remember this.

It is my intention to examine this relationship between government health services and private practice in the various aspects of tuberculosis control.

There are five major divisions of tuberculosis control: case-finding, case-management, rehabilitation, education and prevention.

To some people it appears odd that the finding of any case of disease should be a cause of satisfaction. It represents, after all, human tragedy of greater or less degree. But it is unusual to find this attitude displayed towards communicable disease, and particularly unusual to find it among medical men. However, such an attitude does exist, as one can see from the views of Brailsford (1945), who suggests that widespread attempts at tuberculosis case-finding are unwise unless completely satisfactory facilities exist for dealing with those cases which may be found.

We do not subscribe to these views. Our facilities, while far from perfect, are considerably more adequate—at least in terms of numbers of beds available—than is the case in England, and indeed almost anywhere else. But even if this were not so, it is surely a doctrine of despair to suggest that cases should not be sought or found because the patients cannot immediately be admitted to hospital. Much can be done by simple guidance, by treatment in the home, and by vaccination of those contacts not yet infected, to minimize the spread of infection and disease.

The discovery of cases of tuberculosis depends on a variety of techniques—clinical, radiological, bacteriological and epidemiological. But essentially these techniques are divided into those which arouse suspicion and those which produce confirmation.

It is important economically, and to ensure the best use of trained workers, to assess from time to time the relative productivity of various methods of case-finding.

One feature which distinguishes tuberculosis from most other conditions is the latent period, often quite long, which may intervene between the occurrence of detectable evidence of disease and the onst of symptoms obvious to the patient. This latent period has stimulated the development of mass case-finding techniques, which have arisen in an

[1] Read at a meeting of the South Australian Branch of the British Medical Association on October 25, 1951.

effort to supplement and even to forestall case-finding by the customary processes of clinical examination of persons presenting with symptoms.

Mass case-finding techniques sometimes have a slightly spectacular element, as anyone who has witnessed a mass X-ray investigation of any large American city will know; but experience shows that such surveys may be of very real value in a tuberculosis control programme. Nevertheless, it cannot be stressed too much that there is a far more fruitful field for case-finding among those who actually seek medical advice because of symptoms, and this applies almost as strongly to those with non-respiratory symptoms as it does to those with symptoms directly suggestive of chest disease. For instance, in those general hospitals where all patients admitted have a chest X-ray examination, a surprising proportion of previously unsuspected cases are found in such departments as those of dermatology and radiotherapy.

There are other groups among whom case-finding is more profitable than among the general community. These include recent migrants, women attending ante-natal clinics, and of course contacts of known subjects of tuberculosis.

Let me illustrate these points by reference to recent experience in this State and in Western Australia. Of all new patients referred by private practitioners to the Perth Chest Clinic for investigation because of symptoms, 3·0% proved to be suffering from active tuberculosis.

In our own experience, of 1124 patients referred to the Chest Clinic last year for investigation, 63 or 5·6% proved to be suffering from active tuberculosis. Apparently private practitioners in this State are almost twice as selective as their colleagues in Western Australia.

All persons admitted to the Royal Perth Hospital as out-patients or as in-patients, for whatever cause, have a chest X-ray examination, and of these about 1·0% prove to have active tuberculosis. We have here as yet no experience directly comparable with this. There are some reasons for thinking that our figures may be lower; for example, our tuberculosis mortality rate is slightly lower than in Western Australia, and the proportion yielding a positive Mantoux reaction of students in the University of Adelaide is lower than in a corresponding age group in Western Australia. Even so, it would be surprising if our figures were vastly different. I am convinced that not only is the failure to examine radiologically all comers at the Royal Adelaide Hospital the greatest loophole in our case-finding activities, but it is also a very obvious source of danger to nursing and other staff and to students. Fortunately the deficiency is one which should soon be remedied.

As for migrants, 8·8% of cases of tuberculosis notified in South Australia last year were among post-war migrants. This proportion is very little different from the total proportion of migrants in the community; but because of examination of displaced persons in migrant reception centres in New South Wales and Victoria, any who arrive with active tuberculosis are unlikely to reach South Australia untreated.

I understand that arrangements are in hand for the radiological examination of all ante-natal patients attending the Queen Victoria Maternity Hospital. This will be an important contribution to case finding; it will assist materially in the management of tuberculosis in pregnancy; and it will contribute substantially towards lowering the infant mortality from tuberculous meningitis. For these reasons I believe that X-ray examination of the chest is an essential part of the management of every ante-natal patient, and in a city such as this there is no reason why it should not be done. It is available free through the Chest Clinic, for a modest half-crown at Eagle Chambers, or, of course, in private.

Examination of contacts of known subjects of tuberculosis, by the Chest Clinic, by local officers of health and by private practitioners, contributes substantially to case finding in this State. Last year 1597 contacts were examined by or in association with the Chest Clinic, and 16 of them—1·0%—were found to have active disease.

Turning now to mass surveys, we find that in this community X-ray surveys of the general population and of workers in industry have been unable to produce anything approaching the numbers of cases of tuberculosis found by clinical practice. In industry more than 4000 films have been taken for every new case discovered. In the rural community 2700 films have been required to find each new case; and in the two small metropolitan community surveys which have been completed one subject in each 1800 has been shown to have active tuberculosis. Active tuberculosis has also been found in 5·6% of those referred from doctors, in 1·0% of contacts and in 1·0% of those admitted to hospital in Perth, as against an over-all figure of one in 2500 for mass X-ray surveys. One begins to wonder whether mass surveys are worth the trouble in this healthy community. But it must be remembered that the 50-odd new subjects with active tuberculosis discovered as a result of the State X-Ray Health Survey were all persons who either had not felt it necessary to consult a doctor or visit a hospital, or whose disease remained undetected despite such visits. They were, in fact, generally those in the latent pre-clinical phase which I have mentioned. A number had advanced disease and were clearly unwitting spreaders of infection, while others had minimal infections with a good prognosis but might otherwise have progressed beyond reasonable hope of relief.

At all events, it is clear that our most valuable source of tuberculosis case-finding lies in the surgery of the general practitioner. We can and do provide facilities for following up his suspicions, and we attempt by the examination of special groups and by mass survey methods to find in addition the maximum number of subjects in the latent, pre-symptomatic phase. But these efforts are secondary and ancillary to the discovery of new cases of tuberculosis through the alertness of general practitioners.

The general practitioner has another important role in case-finding: that of advising persons whose survey films arouse suspicion of active tuberculosis. All suspects are referred to doctors nominated by themselves, and we rely on our colleagues in general practice either to carry out the appropriate investigations or to refer the suspect to a specialist in chest disease or to the Chest Clinic.

I think it is fair to say that the system works moderately well, though I am sure it entails a good deal of work by some general practitioners. However, I think I am right in believing that most practitioners and the public would prefer that the present system should continue, rather than that the government health authorities should assume responsibility for the full investigation of all X-ray survey suspects, and report to the patient's doctor only when a diagnosis has been made. I would appreciate any discussion which may arise on this point.

Surveys by Mantoux testing of representative segments of the population produce epidemiological data of the greatest importance. We learn from infection rates in various places and groups what is the impact of tuberculosis on the community, and by serial observations we can determine the trend of the disease and perhaps the effectiveness of control measures.

Mantoux surveys are also a necessary part of any project for the widespread use of B.C.G. vaccine.

In some places, and notably in the State of Minnesota, tuberculin surveys form a very important part of the case-finding programme. School children are tested annually, and an investigation is made of the family and contacts of any child whose response changes from negative to positive. It is claimed by Myers (1948) that the infecting source case is nearly always located within such families.

Such a technique is clearly applicable only in a population in which the proportion of positive reactors to tuberculin is low, and in which the number of children whose response is converted from negative to positive in any year is very small; and, of course, the use of such a technique is completely ruled out by the use of B.C.G. vaccination.

However, the success of the tuberculosis control programme in Minnesota is evidenced by the fact that that

State, which had a mortality from tuberculosis considerably higher than ours fifty years ago, now has one of the lowest mortalities on record, and it is little more than half of ours. There has been a progressive decline in numbers of positive reactors to tuberculin, and Jordan (1947) recently reported from south-western Minnesota that among a school population of over 3000 in his area there was not a single positive reactor.

The medical, as distinct from the social, aspects of case management provide another field for cooperation between those engaged in private practice and those working in State facilities.

In South Australia we are fortunate that there are enough public beds for tuberculosis to allow virtually immediate admission of any female requiring sanatorium care, and to keep the male waiting list down to very small size, both in numbers and in waiting time. This means that there is no necessity for domiciliary care of patients with acute or extensive disease, and beds are usually available for those with minimal disease or pleurisy with effusion. There are, of course, those patients who elect to be treated at home; and provided circumstances are such that this can be done without serious risk of spread of infection, the State is glad to be relieved of the responsibility. We are fortunate in having in Adelaide chest specialists well qualified to advise on the management of patients in this class; and in addition, where facilities for home treatment are satisfactory, and in those stages of the disease where it is appropriate, any advice or assistance which can be given by the staff of the tuberculosis services is gladly offered.

One class of tuberculosis subject frequently treated at home is the patient suffering from pleurisy with effusion. Whatever one's views on the detailed management of this condition, I am convinced that the two mistakes most frequently made are inadequacy of bed rest during and after the acute phase, and insufficient X-ray follow-up in succeeding years. One has seen numerous instances, here and elsewhere, in which patients suffering from so-called primary pleurisy with effusion have been kept in bed for two or three weeks, have returned to work, have perhaps had an X-ray examination of the chest six months later, and have then gone on their way rejoicing, only to return in two or three or more years with extensive pulmonary tuberculosis.

If I make no other point in this paper, I must repeat the plea that every patient suffering from primary pleural effusion remain at rest for a minimum of six months and spend at least the first three months in bed. Thereafter there must be adequate X-ray check for at least five years. Such patients will be paid tuberculosis allowance during their period of treatment, without question and irrespective of the presence or absence of positive bacteriological findings.

I do not propose to discuss in detail techniques of treatment of pulmonary tuberculosis. Members are, I am sure, aware of the general trends—the decreasing use of artificial pneumothorax and the increasing roles of chemotherapy and thoracic surgery. These three trends, I am sure, go hand in hand. Pneumothorax is being used less frequently, because the indications for it are less frequently seen. Bed rest with chemotherapy is undoubtedly more effective than bed rest alone, and patients treated by pneumothorax have always been those in whom bed rest was not producing the desired result. Improvements in thoracic surgery and anæsthesia are limiting the field of pneumothorax at the other end of the scale, and many patients who previously would have been treated with pneumothorax for several years are now having either permanent apical collapse by thoracoplasty, or resection of the affected lobe or lung.

There is much to be learned about chemotherapy in tuberculosis, without even contemplating the introduction of new therapeutic agents. Combined therapy with streptomycin and PAS or thiosemicarbazone has become the established rule, because of better therapeutic response than with any of these drugs alone, and because of pronounced reduction in the emergence of streptomycin-resistant strains of tubercle bacilli.

The toxic effects of streptomycin on the eighth nerve are not now the bugbear that they were in pulmonary tuberculosis. This is mainly because smaller doses have proved just as effective as larger ones. One does not see toxic effects with a dosage of one gramme per day, and there is reason to believe that even this dose is larger than necessary. Intermittent administration has been tried extensively, and doses of two grammes in a single injection three times a week have produced excellent results. However, a single dose of two grammes may produce some toxic symptoms, and we have noted particularly a peculiar circumoral paræsthesia in many of these cases. We are now trying one gramme three times a week.

I think it fair to say that the upper limit of safe dosage of streptomycin has been determined, but the lower limit of effective dosage remains under investigation.

In tuberculous meningitis, in which high dosage is demanded, serious toxic manifestations are unfortunately still seen.

Apart from the few patients who elect to have home care under a general practitioner and a specialist throughout their disease, and the subjects of pleurisy with effusion whom I have mentioned, the main roles of the family doctor in case-management are those of post-sanatorium care and general medical supervision of the tuberculous family. There is often a vast difference between instructions given by the sanatorium superintendent when the patient is discharged and the behaviour which follows. Almoners and visiting nurses may be aware of this, but they cannot always speak with sufficient authority to correct it. We, in the tuberculosis services, must rely on our colleagues in general practice for support in seeing that the recommended regime is carried out, or alternatively, for a suggestion that the advice given does not appear appropriate in the circumstances. Only in this way can we hope to fulfil our functions adequately.

The social aspects of case-management are of vital importance. Without them the medical management of such a prolonged disease would frequently become an intolerable burden. Much is done through the almoner's department of the tuberculosis services, and through the South Australian Division of the National Association for the Prevention of Tuberculosis in Australia, to relieve distress, to provide home help, to care for children, and to assist with housing problems.

Tuberculosis allowances are paid to persons with active disease who are having treatment. The allowances are on a far more generous scale than any other payments made by the Department of Social Services, and are designed to allow sufferers to undertake prolonged treatment without serious hardship, and to enable them to provide for their families on a scale which will serve to protect them from the disease. A married sufferer receives £8 5s. per week *plus* 9s. for each child, in addition to child endowment, and the family may have additional income of £4 per week and unlimited property. There are, in addition, the benefits of the Pensioner Medical Service. I think it can be safely said that these provisions represent a more adequate and realistic attempt to alleviate the distress caused by tuberculosis, and thus to permit its proper treatment, than exists in any other part of the world.

The determination of medical eligibility for tuberculosis allowance is a responsibility of the Director of Tuberculosis in each State. A prerequisite for eligibility is that the sufferer shall have been notified to the Department of Public Health as suffering from tuberculosis. As I indicated when speaking of pleurisy with effusion, it is not necessary that there be bacteriological proof of the diagnosis before notification is made. It is made by any medical practitioner "when he becomes aware" that a person is suffering from tuberculosis. My interpretation of this phrase is that one is so aware when he decides that the patient should cease work and undergo treatment for tuberculosis, on whatever medical grounds this decision may be made.

Rehabilitation.

Rehabilitation of the tuberculous is frequently a problem of the utmost difficulty, though there are many instances in which this is not so—for example, when the patient

returns to his former calling and finds it quite suitable to his altered circumstances.

The rehabilitation division of the Commonwealth Department of Social Services exists to train pensioners of all sorts in fields appropriate to their particular disabilities. Its value in tuberculosis is limited, because it can cater only for those who have a good prospect of full rehabilitation within two years.

The establishment of Bedford industries by the South Australian Division of the National Association for the Prevention of Tuberculosis in Australia, with considerable assistance from the State, and through the untiring efforts of Dr. D'Arcy Cowan, is a most important pioneering venture in this field in Australia. A visit to this combined rehabilitation centre and sheltered workshop is a heartening experience and one which should not be missed by anyone interested in the welfare of tuberculosis sufferers. South Australia's lead has been followed by Western Australia's where there is a similar enterprise in the form of a cardboard box factory in Perth. The Linley Valley Settlement recently opened in the hills some thirty miles from Perth consists of a workshop, a hostel, and several cottages where ex-patients can live with their families and work under sheltered conditions until their working capacity is fully restored.

Those of us who are concerned with rehabilitation are always anxious to have reports from general practitioners about any of their patients who are finding rehabilitation work too difficult or tiring or, of course, too easy.

Efforts to enlighten and educate the public in tuberculosis and its control are made by the Department of Public Health and the Hospitals Department. The role of the general practitioner is, I am sure, to preserve the sense of proportion of the community, so that this or that group of enthusiasts with an approach which is perhaps sometimes a little one-eyed will not succeed in spreading alarm and despondency—or alternatively, undue optimism.

A significant development in education in tuberculosis in this country occurred last week with the constituting of a medical section of the National Association for the Prevention of Tuberculosis in Australia. It is hoped that the section will do a great deal to increase knowledge of and interest in tuberculosis among members of the medical profession, and that it will earn a place of honour similar to that of the Trudeau Society in the United States.

Prevention.

Since the time of Robert Koch, and even earlier, much has been said of the prevention of tuberculosis. Of general measures—diet, housing, working conditions—I need add no more. Of specific immunization with B.C.G. there is much to be said, and indeed much to be discovered. However, certain facts have been established beyond reasonable doubt: first, that properly prepared and administered, the vaccine is harmless; secondly, that it produces in almost every instance an allergy to tuberculin, and that this generally has a duration measurable in years; and thirdly, that it produces in experimental animals a significant degree of protection against subsequent infection with virulent tubercle bacilli. These facts formed a sufficient basis for its extensive trial in man. Unfortunately in many human experiments with B.C.G. controls have been inadequate, and the issue has often been clouded by the presence of other factors which probably contributed to a lowered incidence or mortality from tuberculosis in the vaccinated population. There is also a danger in expecting a benefit in a certain population because a corresponding benefit has been observed in another population living under entirely different conditions. However, despite these reasons for caution, the work of Aronson among North American Indians is most convincing, and a great deal of other experience in the Scandinavian countries, in other parts of Europe, in post-war Japan, and even in our own State, leaves little doubt that the protection afforded by B.C.G. vaccination is very substantial.

Aronson (1948) vaccinated about 1500 children in Indian reservations, and observed them together with a similar number of non-vaccinated controls over a period of eleven years. During that time there were six deaths from tuberculosis among the vaccinated children and 56 among the controls.

In South Australia in the past four years 3500 persons have been vaccinated. Almost without exception they were people particularly exposed to the risk of developing tuberculosis—nurses, medical students, and contacts of known patients. A random sample of the general population, of this size, would during this time have produced four to six cases of tuberculosis. A selected group, exposed to special risk, all giving initially a negative response to tuberculin, and in the early adult age group, would, on the basis of past experience, have produced at least a dozen cases, and probably 20. In fact, in the vaccinated group there has been but one.

B.C.G. vaccine is produced by the Commonwealth Serum Laboratories and distributed through State health departments. The technique of administration is simple. Any person who gives a negative response to tuberculin may safely be vaccinated by a single intradermal injection over the deltoid muscle. For obvious reasons, standard techniques of tuberculin testing and B.C.G. vaccination and a standard record procedure are essential. For these reasons the use of the vaccine is at present restricted, by recommendation of the National Health and Medical Research Council, to those doctors who have been instructed in its use. B.C.G. vaccination is at present carried out at the Chest Clinic once a fortnight, at the Adelaide Children's Hospital for staff only, and also by some ten or twelve doctors in country areas. We are happy to demonstrate the details of technique to any who may be interested, at any B.C.G. session at the clinic. It is hoped in the near future to extend the use of B.C.G. to those children of school-leaving age who give a negative response to tuberculin.

Conclusion.

So, tuberculosis control is a combined operation. The Department of Public Health contributes the mass attack on case-finding. The Hospitals Department provides institutional facilities for investigation and case-management. Both contribute to the B.C.G. programme. Other agencies provide social services, rehabilitation and community education. And private practice contributes the individual approach to all these problems, particularly the three major ones of case-finding, case-management and prevention.

And what effect is this combined operation having?

In the year 1900, with control of tuberculosis in its infancy, the mortality rate from pulmonary tuberculosis in South Australia was 90 per 100,000 of population. In 1950 it was 16·5.

Since the introduction of B.C.G. vaccination in the Royal Adelaide Hospital in 1947, progressive primary tuberculosis in nurses and medical students has almost disappeared.

Because of facts like these, some of us believe that the claim that tuberculosis can be virtually eradicated in our time is not altogether vain.

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Jordon, L. S. (1947), "Results of 15 Years of Tuberculosis Control in Rural Minnesota", *Journal of School Health*, Volume XVII, page 68.

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Reviews.

RECONSTRUCTIVE SURGERY OF THE FACE.

PERWITZSCHKY'S book on reconstruction of the face is printed in German. To prove of interest to Australian surgeons it would need, therefore, to convey a new message in this field. This hope is not fulfilled. Australian plastic surgeons trained in the technique of Sir Harold Gillies and his school would be highly critical of many of the techniques adopted. The crudity of some of the reconstructive work after combined tissue loss is typical of the pre-penicillin era.

The references to resuscitation are brief, to antibiotics absent, to anæsthesia (especially local) extensive, to wound surgery and repair thorough but dated, and to facial and nasal reconstructions reminiscent of Joseph, but dimly illustrated.

The virtues of the book are that it describes personal techniques and cases, is clearly printed and employs that hallmark of the experienced plastic surgeon (the pedicle flap) with skill and understanding.

"JOLL'S DISEASES OF THE THYROID GLAND."

UNTIL the recent war most surgical visitors to London called at the Royal Free Hospital to watch the late Cecil Joll at work on a long afternoon operating list which used to include at least three thyreoidectomies in addition to other major procedures. Visitors would doubt the ability of any surgeon to deal with such a list in one afternoon, and yet by 6 p.m. it was concluded, and they had seen a superb exhibition of manual dexterity, surgical technique and team work. In 1932, when Joll produced the first edition of this book, it was justly hailed as the classical work in British medical literature on the subject, and fifteen years later, Lahey, in concluding the first "Cecil Joll Memorial Lecture" at the Royal College of Surgeons of England, paid tribute to Joll's contribution to the present-day standardization of the surgery of hyperthyreoidism.

It is particularly fitting that this second edition, appearing nearly twenty years later, should have been revised and rewritten by a surgeon closely associated with the author in his later years. In view of the extraordinary advances in the treatment of goitre during this interval, notably Astwood's introduction of the thiouracil group of drugs and the more recent use of radioactive iodine in both the investigation and treatment of thyreoid disease, Mr. Rundle has included a lot of new material on pathology and treatment, also much material from his essay on the "Pathology and Treatment of Thyrotoxicosis", which won the Jacksonian Prize in 1939.

The book has been fully revised and brought up to date, and one notes in particular the review of the latest work on the histopathology of the thyreoid and its relationship to the "thyroid stimulating hormone" of the anterior lobe of the pituitary body, which receives very complete analysis in the opening chapter, also the ocular manifestations of Graves's disease, including the latest work on Mean's "hyperophthalmopathic type", which are ably discussed in Chapter XV.

Recent research is freely referred to throughout and a complete reference list of the appropriate literature is given. Points of criticism are very few, but one cannot but feel a little depressed by the author's statement, made in discussing the relative merits of the various methods of treatment of thyreotoxicosis (Chapter XVII, page 287), that after surgery "about one patient in ten is left with tetany, post-operative myxœdema, paralysis of one or both recurrent laryngeal nerves, or recurrent thyrotoxicosis". Surely modern thyreoid surgery has a lower morbidity than this!

This book is generously illustrated, the photomicrographic reproductions being particularly fine, and the author is to be congratulated on his excellent reediting of a classic which every surgeon dealing with goitres should possess.

[1] *"Wiederherstellungschirurgie des Gesichts"*, by Professor Dr. Reinhard Perwitzschky; 1951. Berlin: Walter de Gruyter and Company. 9½" × 7", pp. 240, with 176 illustrations.

[2] "Joll's Diseases of the Thyroid Gland", by Francis F. Rundle, M.D., F.R.C.S.; Second Edition; 1951. London: William Heinemann (Medical Books), Limited. 10" × 8", pp. 530, with 165 illustrations. Price: 84s.

Notes on Books, Current Journals and New Appliances.

THE WRITING OF SCIENTIFIC PAPERS.

A USEFUL little book for those who wish to learn something about the writing of scientific papers is a small volume by Sam F. Trelease, of Columbia University.[1] It is described as a handbook for students and research workers in all branches of science. The sections of the book deal with "the research problem", "writing the paper", "good form and usage", "tables", "illustrations", "prepublication review" and "proofreading". Generally speaking the advice offered is sound. An author who follows it will leave to an editor only those matters which have to do with the style of the editor's journal or newspaper. The price of the book seems to us to be unduly high.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Your Diabetes: A Complete Manual for Patients", by Herbert Pollack, M.D., and Marie V. Krause, M.S.; Revised Edition; 1951. New York: Paul B. Hoeber, Incorporated. 8½" × 5½", pp. 222. Price: $3.00.

Contains 27 chapters on practical aspects of the subject.

"Approved Laboratory Technic", by John A. Kolmer, M.D., D.P.H., Sc.D., F.A.C.P., Earle H. Spaulding, Ph.D., and Howard W. Robinson, Ph.D.; Fifth Edition; 1951. New York: Appleton-Century-Crofts, Incorporated. 10" × 7", pp. 1192, with 403 illustrations and 28 colour plates. Price: $12.00.

This book is the work of eighteen collaborators.

"Autopsy Diagnosis and Technic", by Otto Saphir, M.D., with a foreword by Ludvig Hektoen, M.D.; Third Edition; 1951. New York: Paul B. Hoeber, Incorporated. 7½" × 5½", pp. 496, with 80 illustrations. Price: $6.00.

First published in 1937.

"Diagnosis and Treatment of Menstrual Disorders and Sterility", by Charles Mazer, M.D., F.A.C.S., and S. Leon Israel, M.D., F.A.C.S.; Third Edition; 1951. New York: Paul B. Hoeber, Incorporated. 9½" × 6½", pp. 598, with 137 illustrations. Price: $10.00.

Intended to serve specially as a guide to the family doctor.

"Angiocardiography", by Charles T. Dotter, M.D., and Israel Steinberg, M.D.; 1951. New York: Paul B. Hoeber, Incorporated. 10½" × 8½", pp. 324, with 635 illustrations. Price: $16.00.

This is Volume XX of *Annals of Roentgenology.*

"International Pharmacopœia", First Edition; Volume I; 1951. Geneva: World Health Organization. 9½" × 6½", pp. 424. Price: 35s.

This is Supplement 2 of the *Bulletin of the World Health Organization.*

"Cancer Cytology of the Uterus: Introducing a Concept of Cervical Cell Pathology", by J. Ernest Ayre, M.D.; 1951. New York: Grune and Stratton. 11" × 8½", pp. 420, with 362 illustrations, some in colour. Price: $14.50.

Based on the study of more than 150,000 genital cell samples from more than 22,000 women.

[1] "The Scientific Paper: How to Prepare It, How to Write It", by Sam F. Trelease; Second Edition; 1951. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 7½" × 5½", pp. 176, with eight text figures. Price: £1 7s.

The Medical Journal of Australia

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All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

MEDICAL RESEARCH IN GREAT BRITAIN.

It is some time since mention was made in these columns of medical research in Great Britain. The reason for this is that a report of the Medical Research Council of the Privy Council for the year 1948-1949 was not issued. The activities of the Council for the period 1948-1950 have now been described in a special document. It takes the place of what normally would have been the thirty-fifth and thirty-sixth annual reports of the Medical Research Council and its predecessor, the Medical Research Committee. The form of the report is different from what it used to be—the document is different for two reasons. The first is that the Council's programme and organization have grown, and the second is that the size of publications of this kind has had to be limited. During the year 1949-1950, the Medical Research Council received as a grant-in-aid £1,216,000 for ordinary expenditure and £319,000 for special non-recurrent expenses. For the year 1950-1951, these sums were £1,316,671 and £295,750. It is interesting to note that the percentage of public funds expended on administration was 4·5. This percentage would be 3·2 if it was calculated on the total ordinary, and non-recurrent, expenditure of public funds. The percentage of public funds expended on general scientific purposes was 0·9; the percentage expended in the National Institute for Medical Research was 22·6; the percentage expended on research units and external staff was 56·5; temporary research grants and training awards accounted for 10·9% of the expenditure, and research on human factors in industrial productivity for 4·6%. During the two years covered by the report, the Council increased its scientific staff organization by the establishment of an Ophthalmological Research Unit and a Spectrographic Research Unit, and by the acquisition of the Antibiotics Research Station. The Council became concerned with the staffing of the Royal Naval Tropical Research Unit at Singapore. It also assumed responsibility for the Ministry of Health Blood Group Reference Laboratory at the Lister Institute of Preventive Medicine, but not as a charge on its own funds. A new Division of Human Physiology was incorporated in the National Institute for Medical Research. At the close of the two years under review the Council's organization consisted of the National Institute for Medical Research, 38 research units (in a few cases with other titles), seven less formally constituted research groups, the Antibiotic Research Station, the Laboratory Animals Bureau, the field research station in the Gambia, and a number of individual members of the staff attached to other institutions. Most of the work was done in the United Kingdom, but investigations by members of the staff were also made in Egypt, in tropical Africa, in Malaya, in the Canadian Arctic, and at sea.

The main part of the report bears the heading "Some Aspects of Medical Research in the Period 1948-1950". We are reminded at the outset that any account of achievements in medical research is bound to be misleading unless the reader bears in mind that virtually every conspicuous advance depends on a mass of unspectacular work, all of which has been indispensable to final results. Among the subjects discussed here is tuberculosis. We read that the death rate from tuberculosis continues to fall in Great Britain, but that the disease is still the main cause of death among men and women of working age. In the report of the Chief Medical Officer of the Ministry of Health in the year 1949, which was published in 1951, it is stated that tuberculosis mortality again declined during that year, the comparative mortality index for respiratory tuberculosis falling a further 7·5% for males and nearly 10% for females, reaching levels of 0·801 and 0·766. Reference is made in the Research Council's report to the effect of streptomycin in tuberculosis. This drug and others in the same category have been only partially effective and the complete cures obtained by chemotherapy in certain other infections have not been matched in tuberculosis. Streptomycin may cause tuberculosis to become quiescent in certain types of cases, but it is seldom sufficient without other forms of treatment. The greater part of the large group of chronic pulmonary tuberculosis is practically unaffected by chemotherapy. Unfortunately, when streptomycin is given to a tuberculous patient, the tubercle bacilli often become resistant before very long to the action of the drug. During the period covered by this report, the most important chemotherapeutic advance in this field has been the discovery that the development of resistance to streptomycin can be minimized by the use of *p*-aminosalicylic acid (PAS). The work of the Council's Tuberculosis Research Unit contributed to this discovery. In the report of the Council for the years 1945-1948, it was shown that rigorously controlled clinical trial of drugs in tuberculosis was feasible and could be carried out if a large number of different centres accepted central coordination. The precision with which it has been possible to define the value and limitation of the combined use of streptomycin and PAS is, we are told, due in no small measure to the adoption of this technique. Animal work has been carried out in Great Britain on the use in tuberculosis of synthetic drugs such as the thiosemicarbazones, developed in Germany, and the results have suggested that the benefit from their use may be more than offset by their toxicity. A cautious attitude is displayed towards the use of B.C.G. vaccine. It is stated that the design of mass efforts in

many countries with B.C.G. has made it impossible to judge the degree of benefit likely to accrue in the years to come. In Great Britain, B.C.G. vaccination is at present being offered as a routine only to persons known to be in contact with tuberculosis, for example, hospital nurses and children in tuberculous families. It is, we read, important to decide as soon as possible, whether under the conditions existing in Britain extension to the general population of the use of B.C.G. vaccine would give a substantial degree of protection. Unless B.C.G. can do this, its general use might do more harm by giving a false sense of security and by distracting effort from the only certain preventive measure, namely, the eradication of sources of infection. In Australia it is believed by many experts that the value of B.C.G. has been proved and that its general use is justified. Reference is made to the discovery that large doses of cortisone aggravate tuberculosis in experimentally infected mice, and that in infected guinea-pigs this substance also diminishes the tissue response to the proteins of the bacillus, whereas the thyroid hormone, thyroxin, increases it. It is thought that extension of these studies may provide fundamental information on how the body is capable of resisting tuberculosis. In the section on virus diseases, reference is made under the heading of "Influenza" to the work of L. Hoyle on the life cycle of the influenza virus. This work "suggests that after injection into the fertile egg, the virus may break up into five particles which reproduce themselves before coming together in some way to reconstitute the normal virus bodies". We are told that it is too early to say how far the suggestion is correct, but that it fits in well with other work that is being carried out in the United States and in Australia on the fascinating problem of "recombinations". It has been found that, when a cell is infected simultaneously with two suitable strains of virus possessing different properties, variant forms may sometimes be recovered endowed with properties different from those possessed by either of the parent forms. In regard to the common cold, it is stated that the work on the epidemiology which was reported in the last report has been continued. It has been found in the community being investigated that school children experience about three times as many colds as did adults living in a household without school children; moreover, the presence of school children in a household approximately doubled the number of colds experienced both by adults and by children under five years of age. In regard to poliomyelitis, reference is made to the view that among the factors which may contribute to the onset of paralysis, an important one may be any local "injury" to the tissues. This injury, it is thought, may range from an incident as drastic as a bone fracture or a surgical operation, to nothing more obviously damaging than unusually prolonged or severe physical exercise. Among these so-called injuries have been protective inoculations against diphtheria or against diphtheria and whooping-cough. Observations of this kind were reported from Australia. At the request of the Ministry of Health, the Medical Research Council has set up a committee to study in greater detail the association between inoculation and paralysis, to estimate the risk of the double event under varying circumstances, and to recommend any additional precautions or adjustments in existing practice that may be desirable. Reference is made to "Q" fever. Its early history is mentioned and some details are given of an outbreak of the disease in London in July, 1949. It is pointed out that the recognition of "Q" fever in Britain is of interest not only in itself, but also because it marks a further step in the differentiation of what have been called pyrexias of unknown origin. During the past thirty years or so, virus disease, undulant fever, rat-bite fever, lymphocytic choriomeningitis and psittacosis have been disentangled from the group, but the separation of "Q" fever has narrowed it down still further. The future, we read, seems to lie clearly with the virologist, whose task it will be to discriminate similarly between the numerous catarrhal diseases of the respiratory tract and the no less confusing group of inflammatory disorders of the central nervous system. In this regard, the interest of virologists the world over will centre in the recent investigation reported from the Walter and Eliza Hall Institute in Melbourne of Murray Valley encephalitis. The discovery by Phillip Hench of the action of cortisone is described as a spectacular discovery in medical research during the two years under review. Emphasis is laid on the fact that cortisone and ACTH do not cure rheumatoid arthritis; they suppress its symptoms which may return soon after treatment has been stopped. Serious symptoms such as mental disturbances, the flaring of quiescent tuberculosis, and the development of heart failure due to salt and water retention have been reported following the use of these drugs. The conclusion is that at present treatment with them should be undertaken only where there are facilities for full laboratory control, and that they cannot safely come into general use until further research has been completed. The biological aspects of atomic physics are discussed. In regard to tracer elements in medical research, reference is made to the accurate method of measuring thyroid efficiency. A single dose of radioactive iodine is given by mouth and later radiation measurements of the iodine in the urine are made; this has now become routine practice in several institutions. In regard to the use of radioactive isotopes, we are told that it is still too early to assess the ultimate scope of isotope therapy. Clinical work of this kind can be carried out only under carefully defined conditions at suitably staffed and equipped centres. The chief consideration must always be to protect the patient from any immediate damage to the sensitive tissues, and from the more remote but by no means negligible risks of genetic damage, or of actual production of cancer. Radioactive isotopes should therefore not be used in non-malignant conditions unless as in certain cases of toxic goitre, there is no safer alternative, and great caution must be exercised when giving them in therapeutic doses to patients of either sex in the reproductive period of life, or to any patient with an expectation of life exceeding five years. The only other subject to which we would draw attention is that of under-nutrition. Investigations were made in Germany into the question whether under-nutrition in itself interferes with the ability to produce antibodies to specific infections. A group of undernourished and normal volunteers were subjected to a series of powerful but innocuous stimuli of which they could have had no previous experience. The results showed that healthy people do produce antibodies rather more efficiently than those who are undernourished; the differences, however, were found

to be small, and it is not thought that deficiencies of this order can have played a major part in promoting the spread of epidemic diseases such as typhus fever, typhoid fever, and scarlet fever in times of famine. The effect of undernutrition on the activity of some of the serum enzymes was investigated. It was found that the activity of cholinesterase fell during undernutrition and rose again with recovery. This finding has been made the basis of a test for undernutrition. It is pointed out that the psychological changes induced by undernutrition are among its most important manifestations—many of the signs and symptoms, such as sweating, breathlessness, giddiness and insomnia, commonly regarded as physical effects, may be psychological in origin.

From the foregoing references to the activities of the Medical Research Council of Great Britain, it will be clear that investigations have ranged over the whole face of medical science. It is important that medical practitioners in this country should know what is being done in the older centres in Great Britain. They will then be in a position to appreciate more easily the scope of investigations which should be fathered by our own National Health and Medical Research Council. It is unnecessary to point out that research in Great Britain or in Australia or in any other country must be regarded as a common effort directed towards a common end—the discovery of the cause of disease and of its prevention. It is important, too, that those in high political places in this country who control the expenditure of public money should appreciate the value of what is done and the fact that Australia must take her place, her full place, in the common effort.

Current Comment.

VOLKMANN'S ISCHÆMIC CONTRACTURE IN THE LEG.

THE term Volkmann's ischæmic contracture is usually applied to a contraction deformity of the upper limb, in accordance with the original description by von Volkmann in 1896 of a lesion at that site. Apparently following the prototype, some authorities, at least by implication, limit the term to the upper limb. Beattie and Dickson, in their text-book of pathology, are content to close their description of "this deformity of the upper limb" with the statement: "Analogous lesions in the lower limbs have been described." There seems to be no good reason for limiting the term to one site, and absence of discussion on the point can probably be attributed to the rarity of reports of "analogous lesions in the lower limbs". It may be that the actual incidence is greater than the number of reports suggests; S. A. Thomson and L. J. Mahoney, of Toronto,[1] quote the comment made by Parkes in 1945: "It is perhaps unfortunate that attention has been so focused on this region [the flexor muscles of the forearm] that it is probable the condition has frequently passed unrecognized in other muscle groups." Thomson and Mahoney state that they have been struck in their clinic at the Hospital for Sick Children, Toronto, with the "menacing occurrence" of Volkmann's ischæmia in the lower extremity and with the almost complete lack of reported examples from other centres. In their experience over a period of seventeen years 42 cases of ischæmia were observed; 29 were old or well-established cases of contracture and 13 were cases of acute ischæmia. Of the old cases 20 involved the arm and nine the leg, all the second group as complications of a fractured femur. Of the acute cases, four involved the arm and nine the leg; eight followed simple fracture of the upper half or third of the femoral shaft and one followed complicated rotation osteotomy of the tibia. It may be noted for comparison purposes that during the period of seventeen years under discussion, 1233 patients with fractured femur were admitted to the Hospital for Sick Children. The routine method of treatment was by skin traction applied to the leg in a Thomas's splint.

Thomson and Mahoney state that in all their cases pain has been the important diagnostic feature. It is usually in the central calf area and is of the deep boring type which is difficult to control with sedatives and is not much influenced by the release of bandages. It is immediately accentuated by passive dorsiflexion of the foot. A sensation of numbness is soon recorded in the toes and foot and may extend up to the painful calf; this is associated with a stocking type of anæsthesia. However, the painful calf area remains exquisitely tender to pressure. Also noted on the skin over the calf is an irregular red raised erythematous area with bleb-formation soon superimposed. The calf becomes swollen, as does the foot, with variations in colour and temperature. Arterial pulsation may be normal, at least for some time. Movement of the foot and toes becomes impaired at an early stage; total paralysis follows in a few hours, and the foot rests in a position of equinovarus. From the diagnostic and prognostic points of view it is well to note Thomson and Mahoney's comment: "The early signs and symptoms in the calf are ominous, whereas the early vascular signs of a good tibial pulse, colour and temperature are often misleading."

A certain amount of light is thrown on the management of the condition by this paper. Muscle decompression with or without arterial exploration has proved of no value in Thomson and Mahoney's experience. They consider that early recognition of the condition and radical treatment are imperative. Temporary paralysis of the lumbar sympathetic outflow, as by long-lasting high spinal anæsthesia, has been found effective, as has paravertebral sympathetic block, which has the advantage of depressing the blood pressure less than spinal injection. Transfixion of the calcaneus with a Kirschner wire for traction has the advantage of eliminating all bandages, suspending the tender calf and preventing an equinovarus deformity.

Ultimately, satisfactory measures for both prevention and treatment of Volkmann's ischæmic contracture will probably come only when the ætiology and pathology of the condition are understood. Writing elsewhere in the same journal, D. Ll. Griffiths points out that the biggest unsolved mystery about the condition is why the ischæmic muscle shrinks. He considers that it is the dead muscle which contracts and, in shrinking, imposes deformity upon the limb. It is time, he states, that our attention was diverted a little from the ischæmia to the contracture. If we could discover the mechanism of shrinkage we could do something to prevent it in cases in which ischæmia persists despite treatment. He refers to Thomson and Mahoney's use of skeletal traction through the calcaneus when the calf is involved and quotes the opinion of H. J. Seddon that radical excision of the infarcted muscle is a hopeful procedure in the forearm if the excision is performed early enough. General agreement has apparently been reached, Griffiths states later, that major arterial obstruction is necessary for the production of Volkmann's contracture. Primary traumatic arterial spasm is important, but probably a less common cause of the obstruction than are organic blocks. Pointing out that the only known factor certainly important in its production is traction, Griffiths stresses the view that traction on the brachial artery in front of the elbow during manipulation of a supracondylar fracture is to be avoided at all costs, as is traction on any severely injured lower limb. One of the most disturbing instances in Thomson and Mahoney's series is the case of the child in a "gallows" type of extension who developed ischæmia in the uninjured limb. Uncommon as Volkmann's ischæmic contracture is in most practices, this and many other features of the Toronto experience suggest the need to stop and think sometimes even about well-established procedures.

[1] *The Journal of Bone and Joint Surgery*, August, 1951.

Abstracts from Medical Literature.

MEDICINE.

Clip-on Electrocardiographic Electrodes.

E. SIMONSON (*American Heart Journal*, September, 1951) describes how limb electrodes for electrocardiography may be simply made from bicycle clips (known as pants clips in the United States of America). It is stated that these may be affixed to the patient in about half the time taken to fit the conventional rubber strips.

Herpangina, or Viral Ulceration of the Throat.

R. H. PARROTT *et alii* (*The New England Journal of Medicine*, August 23, 1951) describe clinical studies of a common summer illness of children, characterized by fever, sore throat and vesicular or ulcerated lesions in the anterior faucial pillars or soft palate. The illness is identified as herpangina, originally reported by Zakorsky in 1920. Viruses similar to those classified among the Coxsackie viruses of group A were recovered from the majority of the patients.

The Effects of Tubing Bore on Stethoscope Efficiency.

M. B. RAPPAPORT AND H. B. SPRAGUE (*American Heart Journal*, October, 1951) have conducted instrumental tests to determine the effects of variation of the calibre of the tubing on the acoustical efficiency of a stethoscope. They have found that tubing one-eighth of an inch in bore produces a stethoscope of much higher efficiency than tubing with the customary calibre of three-sixteenths of an inch, except in a range of sound-wave transmission that has practically no application to cardiac auscultation. The bore of the metal parts should be the same as that of the rubber tubing.

The Impropriety of Strict Bed Rest in Heart Failure.

S. A. LEVINE (*American Heart Journal*, September, 1951) marshals clinical and physiological evidence to support the view that strict bed rest is deleterious to patients with congestive heart failure. He states that there is abundant reason to believe that, if heart failure is present, the heart obtains more rest if the patient sits in a chair with the feet down than if he lies in bed, even if the head and back are elevated. The author has treated 70 patients with acute coronary thrombosis by having them placed in chairs for varying and increasing periods of the day, beginning within a week of the attack, and has found the results encouraging and the method possessed of physical and psychical advantages over treatment by strict bed rest.

A Rocking Bed for Respiratory Paralysis.

F. PLUM AND G. D. WHEDON (*The New England Journal of Medicine*, August 16, 1951) have conducted experiments in the use of rocking beds in the treatment of patients suffering from respiratory paralysis. In the acute stage of poliomyelitis the "iron lung" type of respirator was found the more satisfactory, but with convalescent patients who could remain out of the respirator for five minutes or so the rocking bed permitted sufficient pulmonary ventilation and facilitated nursing care and enabled several patients to overcome feelings of dependence on the respirator. The bed was rocked through an arc of about 40°, which was the maximum that could be comfortably tolerated for long periods, and the rate of rocking was from 16 to 24 cycles per minute.

A Low-Cholesterol Low-Fat Diet in Coronary Atherosclerosis.

L. M. MORRISON (*American Heart Journal*, October, 1951) has followed for three years 100 patients, all of whom had recently recovered from infarction of the heart. Fifty of the patients took a diet with a low content of fat and cholesterol, while 50 took the same food as they had taken before their illness. Fourteen of the former group died during the observation period, but 30 of the control group died. The patients taking the diet of low-cholesterol and low-fat content lost about 20 pounds in weight during the observation period, whereas the weight of the patients in the control groups was not significantly altered. The author suggests that a diet poor in cholesterol and fat for the treatment of coronary atherosclerosis merits further controlled study. The diet in question lists as "foods to be avoided" cream soups, glandular meats, pork and fat meats and fish, oysters, roe, whole milk, cream, rich cheese and cheese spreads, excessive butter and butter substitutes, egg yolks, pies, frozen creams, rich cakes, salad dressings, oils, suet, fats, gravies, olives, nuts and avocadoes.

Prognosis of Angina Pectoris and Coronary Occlusion.

LOUIS H. SIGLER (*The Journal of the American Medical Association*, July 14, 1951) discusses the prognosis of *angina pectoris* and coronary occlusion, and presents a statistical study of 1700 patients suffering with these conditions, observed over a period of twenty-five years. The ratio of males to females was about 3·2:1·0. The author states that the pathology of the underlying disease is either arteriosclerosis or atheromatosis, and in some cases intra-arterial wall hæmorrhage. These cause gradual narrowing and finally complete obstruction of one or more branches of the coronary arterial tree with or without a fresh thrombosis. In many cases the first clinical manifestation is either an acute episode of coronary occlusion or sudden death; autopsy may reveal extensive coronary disease. For this reason, the actual prognosis of coronary disease from its early development to death cannot be estimated. In a great many cases the symptomatology of coronary disease, expressed mainly in the anginal syndrome, does not progress uninterruptedly from its inception to the end. There are periods of remission or quiescence and exacerbation or crisis. It is likely that in many cases during the period when the anginal syndrome is present, closure of vessels is taking place, even though the classical criteria of coronary thrombosis are absent. In many cases of uncomplicated *angina pectoris*, the patients have been found to have old occlusions of one or more coronary arteries. The typical picture of coronary occlusion was absent, as occlusion was gradual and collateral circulation developed. Because of the periods of remission of the anginal syndrome, extreme care is indicated in taking a history to determine the exact time of onset of symptoms. The mode of onset in this series was either as an acute episode, or in a slowly progressive manner. In half of the cases, the onset was acute in the classic form of coronary occlusion; this occurred more frequently in males. Certain findings suggest that the chances of survival are greater if the onset of the disease occurs earlier in life. Of the 1208 patients with coronary thrombosis in this series 72·6% had no clinical evidence of coronary disease before the first attack of coronary occlusion. In 330 cases in which symptoms preceded the first attack of coronary occlusion, the duration of preceding symptoms varied greatly; it averaged 3·9 years for males and 3·4 years for females. The average duration of life of the 423 patients who eventually died was only 3·8 years for males and 3·1 years for females; for the 785 patients who were still alive, it was, to date, 4·9 years for males and 4·5 years for females. It was observed that 83·1% of males and 81·5% of females had remained alive for five years or longer, and 10·7% of males and 11·4% of females for ten years or more. One patient was still alive thirty years after an attack of coronary occlusion. The over-all average age at onset for the entire series was 55·8 years, the youngest patient being aged twenty years and the oldest ninety-three years.

The Effects of BAL in Hepato-Lenticular Degeneration.

J. A. CUMMINGS (*Brain*, Volume LXXIV, Part I) states that it has been shown that in cases of hepato-lenticular degeneration there is an excess of copper in the tissues and in the urine. There may also be an excess of amino-acid nitrogen in the urine. It was found that in patients who improved with BAL therapy, urinary excretion of copper, but not of amino-acid nitrogen, was lowered. Experience with these cases suggests that it may be beneficial to give short courses of BAL. It is too early to state how long the improvement will last.

Mucoid Impaction of the Bronchi.

R. R. SHAW (*The Journal of Thoracic Surgery*, August, 1951) uses the term "mucoid impaction of the bronchi" to designate a clinical entity, not hitherto clearly described, resulting from a localized accumulation of inspissated mucus in the bronchi of persons suffering from asthma or chronic obstructive bronchitis. The impaction occurs usually in a second-order branch bronchus distally to a bifurcation and grows in size, layer by layer, until finally it dilates the bronchus to many times its normal diameter. As it enlarges it compresses the neighbouring branch bronchus so that it also accumulates a mucoid mass which enlarges in the same manner. The dilated bronchial walls become infected and degenerate, and a non-putrid abscess may result. The symptoms and X-ray appearances are variable. Usually there is a febrile episode with fever initially high, responding well to chemotherapy and then persisting with a slight daily

rise of temperature. Diagnosis of the disease from pulmonary neoplasia may be impossible without thoracotomy, at which operation segments of lung destroyed by suppuration may be removed.

Exfoliative-Cell Diagnosis of Central Nervous System Lesions.

WILLIAM R. PLATT (*Archives of Neurology and Psychiatry*, August, 1951) describes techniques for examination of desquamated products extruded into spaces containing cerebro-spinal fluid and taken from the lumina of cerebral cysts. He states that this examination is a useful adjunct to other ancillary neurological investigations. It is often helpful in the differential diagnosis of neoplasms from congenital, degenerative and inflammatory states.

Divergence Paralysis with Increased Intracranial Pressure.

MAX CHAMLIN AND LEO M. DAVIDOFF (*Archives of Ophthalmology*, August, 1951) review the subject of divergence paralysis and report two cases. They have encountered five cases and have collected ten others from the literature, and in all there was a raised intracranial pressure. In eight cases there was a focal lesion in the posterior fossa, and in ten improvement followed relief of the increased intracranial pressure. The authors conclude that in cases of known increased intracranial pressure with diplopia, if definite paralysis of one or more muscles is not easily found, divergence paralysis must be suspected and tested for. Conversely, if divergence paralysis is encountered, increased intracranial pressure should be suspected and sought, even in the absence of frank papilloedema.

Photophobia: Mechanism and Implications.

JAMES E. LEBENSOHN (*American Journal of Ophthalmology*, September, 1951) distinguishes between dazzling and photophobia. He states that the cause of dazzling is generally inadequate adaptation, and in contrast to photophobia it is not accompanied by blepharospasm and lachrymation. The integrity of the fifth nerve is essential to photophobia. The author has evaluated the variations in intensity of photophobia by means of rotating polaroid filters. He states that photophobia may accompany a lesion in any area supplied by the ophthalmic division of the fifth nerve. Whatever the cause, true photophobia is based on iritic constriction to light. The light reflex becomes painful after the iris has been sensitized by trigeminal axon reflexes producing vasodilatation and hyperalgesia. Photophobia, regardless of ætiology, is measurably alleviated by adrenaline, "Neo-Synephrine", mydriatics and inhalations of trichlorethylene.

ACTH and Respiratory Depression.

R. W. KEETON *et alii* (*The Journal of the American Medical Association*, June 16, 1951) describe two cases of dramatic respiratory symptoms induced by the withdrawal of ACTH. They state that prolonged treatment with ACTH may cause effects due to excessive release of corticoids or to action on the anterior pituitary lobe and adrenal cortex. Their first patient, a man, aged forty-eight years, with pulmonary fibrosis, *cor pulmonale* and cardiac failure, was dyspnœic and had coughing spells. The administration of 20 milligrammes of ACTH thrice daily produced improvement, and the hormone was continued for two months. On its withdrawal, dyspnœa and cough returned, and the patient became depressed, hysterical and exhausted. The hormone was again administered with improvement. One month later the hormone was suspended again, with depression and dyspnœa; the patient responded to resumption of ACTH, and eventually a dose of 30 milligrammes daily in divided doses was given. The second patient had chronic bronchitis and emphysema; he gave the same response to ACTH, and the drug had to be withdrawn very gradually. The authors suggest that pituitary exhaustion was the cause of the relapse after withdrawal, whereas adrenal exhaustion follows withdrawal of cortisone.

Fundus Changes in Juvenile Diabetics.

LAWRENCE T. POST AND ARTHUR W. STICKLE (*American Journal of Ophthalmology*, August, 1951) present an analysis of their findings from examination of the fundi of 59 juvenile diabetics. They found that poor control of the disease and its duration were important factors in development of retinopathy, kidney damage and poor general health Examination showed that 54% of the patients studied had retinopathy. The authors classified the retinopathy as minimal, moderate and advanced, there being a definite increase in the severity and incidence of retinopathy with the duration of the diabetes. There was also a close correlation between retinopathy and the general physical condition and amount of kidney damage. Capillary fragility increased with an increase in diabetic retinopathy. Capillary aneurysms and small round hæmorrhages were the first changes to be seen, and they were usually found in the area between the superior and inferior temporal vessels. The authors conclude that in juvenile diabetics examination of the retina is a fairly reliable method of determining the amount of kidney damage and the condition of the vascular system in general.

Acne Vulgaris.

G. C. ANDREWS *et alii* (*The Journal of the American Medical Association*, July 21, 1951) describe the treatment of *acne vulgaris* with antibiotics and œstrogens. The results obtained were thought to be better than those obtained with X rays. The authors treat ordinary simple acne with a diet of low fat content, vitamin *A*, diethylstilbœstrol, expression of comedones and local remedies, such as *lotio alba*, sulphurated lime solution, and treatment of the scalp. They state that pustular, nodular and cystic acne is due to sensitization to *Staphylococcus albus* and may be due to foci of infection, such as pyelonephritis, sinusitis and infected teeth, tonsils or appendix. For this type of acne the authors recommend terramycin, aureomycin, "Chloromycetin" and sulphonamides, in addition to removal of the focus of infection. They state that these staphylococcal conditions respond to different antibiotics, penicillin, aureomycin *et cetera*, and the cocci should be investigated in relation to each of the antibiotics. In their experience sulphadiazine, in a dosage of two grammes daily for two weeks, then 1·0 to 1·5 grammes daily for eight or ten weeks, often gave good results in pustular cases. Diethylstilbœstrol, 0·25 milligramme, was given daily to male patients or to female patients throughout the menstrual cycle, unless changes in the cycle occurred, in which case the hormone was given only during the first two weeks of the period. Some danger of affecting the sex development of young boys and girls is admitted, and it is stated that short courses of therapy are to be preferred. Other methods of treatment were used in the authors' series, but they strongly favour the use of sulphadiazine, antibiotics, œstrogens and diet. They compare 253 patients treated by X rays with 384 patients treated with sulphadiazine, removal of foci of infection, diet, vaccines, crude liver extract, vitamin *B*, iron, arsenic, vitamin *A*, vitamin B_{12} and other methods.

Status Epilepticus with ACTH.

A. DORFMAN *et alii* (*The Journal of the American Medical Association*, May 5, 1951) describe the occurrence of *status epilepticus* in three patients coincident with the use of ACTH. One child, aged thirteen years, was treated for dermatomyositis with 20 milligrammes of ACTH given intramuscularly four times a day. Considerable improvement in his skin and general condition was noted. Later a state of mental confusion developed, and he had a series of epileptiform convulsions. He recovered from the acute illness, but after a year has not regained normal mentality and continues to have convulsions preceded by impulsive destructive behaviour. The second child, aged six years, was treated for recurrent rheumatic fever with 60 milligrammes of ACTH for four days, then 40 milligrammes for ten days. She lapsed into convulsions and coma. The electroencephalogram had not returned to normal in one month. The third child, aged ten years, had a macular rash with fever and general pains. *Periarteritis nodosa* was diagnosed by skin biopsy. ACTH was given in doses of 20 milligrammes four times daily for three days. After three days, 60 milligrammes were given daily for two weeks. Improvement occurred in relation to temperature and rash. A dose of 80 milligrammes daily was again given for two weeks, and then 60 milligrammes daily for four weeks to maintain the remission. Acne and round face developed. The blood pressure rose. The dose was gradually reduced to 20 milligrammes a day. On the ninety-second day of treatment the child became irrational and had a major convulsion; *status epilepticus* developed, followed by coma. From this he recovered in a few days and became normal mentally, but his electroencephalogram continued to show pronounced abnormality. The authors state that the hazards of salt retention, disturbances of carbohydrate metabolism, potassium deficiency and hypertension have in general been less severe than was anticipated, when cortisone and ACTH have been used. In the cases reported, one child was mentally abnormal before treatment, but the significance of convulsions in the other two cannot be overlooked.

Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

VI.

THE APPLICATION OF THE ELECTROCARDIOGRAPH IN GENERAL PRACTICE.

AT the outset let me make it quite clear that this article is in no way a synopsis of the subject of electrocardiography. In it will not be found a detailed exposition of any of the intricate problems of this subject, for it is written solely with the idea of advising the general practitioner in which type of case he is likely to obtain from this method of examination some assistance in clarifying the clinical picture, in controlling treatment, or in assessing the prognosis.

What Does the Electrocardiograph Do?

The electrocardiograph records changes in the electric potential which accompany activity or injury of the myocardium. It shows the site of origin of the impulse which causes the heart to contract. It gives evidence concerning the conduction of this impulse and then of the consequent myocardial activity.

The form of the electrocardiogram is altered, not only by physical and chemical changes in the cardiac tissue, but also by vagal and sympathetic nervous impulses, as well as by the position of the heart. What the electrocardiograph does not do is demonstrate the presence of failure, nor does it measure the degree of muscular damage, although it may assist in its assessment.

When will this Examination be of Most Use to the General Practitioner?

In country areas it may sufficiently often be of use to make it worth his while to take his own tracings, a not very difficult technical procedure. However, with the number of leads now in use, the work is time-consuming, while in addition practice in interpretation is essential. It might well, on occasion, repay the practitioner for his trouble by enabling him to determine the type of irregularity or of paroxysmal tachycardia, or the presence of an infarct or myocardial ischæmia, but, as is so often the case with special methods of investigation, it is most likely to give him an unequivocal answer only in those cases in which he should have arrived at the correct conclusion by the exercise of his clinical acumen; whereas in the difficult borderline cases the difference between the normal and the abnormal can be recognized only by judgement developed from constant practice in interpreting large numbers of records. So in the case in which he most needs it he may find help most difficult to obtain. Actually the expert finds his greatest trouble in distinguishing the abnormal from the variations of the normal. Perhaps one might cite the *Q3 T3* pattern of posterior infarction which is always liable to prove a trap. Taken by and large, the average busy practitioner will find it advisable to obtain an opinion from a specialist, but he cannot expect this to be adequate unless the consultant is given the opportunity of being conversant with the history and clinical findings.

Although some information is likely to be obtained from the electrocardiogram in all cardiac conditions, it is only in certain cases that it will be of sufficient practical importance to the general practitioner to warrant the extra investigation. As this article deals with the practical application, let us therefore consider in what circumstances he will find assistance worth while. I say assistance advisedly, as the electrocardiogram only rarely provides the most important evidence, for it is usually only one piece of the jig-saw which must be fitted into its proper place in the picture as a whole.

Conditions in which the Electrocardiogram May Prove Worth While.

Perhaps one of the most useful roles of the electrocardiogram from the practical point of view, though a minor one scientifically, is to produce a normal record as evidence in black and white to help convince the cardiac neurotic that his heart is normal. On the other hand, among abnormal conditions, evidence relating to coronary artery disease is almost certainly the most important field of investigation. The indications may be considered under three headings.

Diagnosis.

In diagnosis we may use three subgroups:

Abnormalities of Rhythm.—I have no intention of discussing all the innumerable curious components of this group. The common conditions include sinus arrhythmia, extrasystoles, auricular fibrillation and flutter, and paroxysmal tachycardia. The main reason why it is so important to be quite sure of the cause of the irregularity is that there is a vast difference in the significance of auricular fibrillation and flutter on the one hand and that of mere sinus arrhythmia and/or extrasystoles on the other. Clinical examination usually suffices to make the diagnosis, for deep breathing will make sinus arrhythmia more obvious while premature beats or extrasystoles usually disappear with a more rapid rate following exercise. However, on occasion the presence of both conditions simultaneously causes an irregularity very difficult to distinguish from auricular fibrillation until the electrocardiogram is taken.

The irregularity of auricular fibrillation is usually obvious unless the rate is slow, in which case speeding up usually gives the answer, but where there is any doubt it is important to resolve it by means of a tracing.

On clinical examination frequent *a* waves in the jugular vein may raise the suspicion of flutter, even when the rhythm is regular. Irregular ventricular response to auricular flutter usually calls for an electrocardiogram to differentiate it from fibrillation *et cetera*. When pressure on the carotid sinus abruptly halves the rate in tachycardia it suggests flutter, although sometimes the pressure only increases the irregularity, but a lead taken from the second right intercostal space usually shows the flutter waves clearly in the long gaps.

Paroxysmal tachycardia can usually be diagnosed on the history alone by its abrupt onset and offset.

The differentiation between auricular and ventricular types is important because of the difference in both treatment and prognosis.

Disorders of Conduction.—Conduction through the auricles, the *A-V* bundle and its branches and through the walls of the ventricles may be impaired by many conditions, but the two most common groups of persons affected are: (*a*) young people with acute rheumatism or infections, (*b*) older people with coronary artery disease.

As we are viewing the subject from the practitioner's point of view, it is obvious that in some of these cases a block will be missed, because, although some myocardial involvement may be recognized, there is no urgent indication for an electrocardiogram. There are occasions, however, when it is obviously important to determine whether a faint turn or blackout is caused by heart block or by vasovagal means.

When the Stokes-Adams syndrome is suspected of being the cause of faints it is important to have a tracing taken during an attack because sometimes the symptoms are caused by the onset of ventricular fibrillation during the block, which has an important bearing on the treatment to be administered.

Partial heart block with delayed conduction through the bundle of His, which in the recording is indicated by a long *P-R* interval, may be guessed at on clinical examination if the auricular sound is heard a little before the ventricular first sound, but this is rare and only very occasionally is the cause of a gallop rhythm. If the block is greater there may be dropped beats which may arouse suspicion if they are not misinterpreted as being the result of premature beats and compensatory pauses.

These may sometimes be recognized if they can be eliminated by atropine, but usually the electrocardiogram is required for the diagnosis. When the pulse rate is 40 per minute or thereabouts, heart block is suspected; but although occasionally the *a* waves in the jugular pulse may indicate this mechanism and differentiate it from sinus bradycardia, it is frequently necessary to invoke the aid of the electrocardiograph. Sinus bradycardia due to vagal impulses will sometimes be found to be the cause, but, on the other hand, complete heart block may be present in spite of a more rapid ventricular rate, in which case it may not be suspected.

Splitting of both first and second sounds may suggest the presence of a bundle branch block, especially a wide splitting of the pulmonary second sound which often occurs in right bundle branch block. However, proof comes only from the chest leads of the electrocardiogram which will show whether the block is mainly in one branch or the other, but this latter information is usually, although not always, of academic interest.

Myocardial Lesions.—It must be borne in mind that coronary sclerosis and coronary occlusion in themselves do not alter the electrocardiogram. The changes recorded are due to myocardial damage caused by these conditions with either ischæmia or actual infarct formation, the former tending to flatten and invert the *T* waves, while the latter is apt to bring about the prominent *Q* wave due to death of muscle, elevation of *S-T* due to the current of injury, and subsequent inversion of *T* due to ischæmia.

It would be interesting if one could determine which patient was most likely to develop a coronary occlusion. A possible half-way stage between ischæmia and occlusion, which has been termed coronary insufficiency, shows depression of the *S-T* segments. Although an infarct may be present and yet not cause obvious electrocardiographic changes, it is usual to find some indication if an adequate number of chest, unipolar, and augmented limb leads are recorded.

An infarct involving the full thickness of the ventricular wall will, of course, make itself fairly obvious, while a smaller infarct surrounded by more or less normal myocardial tissue will be more likely to be obscured. In cases of doubt an electrocardiogram will sometimes be necessary to distinguish coronary occlusion from perforated ulcer, cholecystitis, pancreatitis *et cetera.*

The right choice of leads may produce fairly accurate localization of the infarct, but this is frequently of scientific interest rather than of practical value. However, some idea of the extent of the infarct may sometimes be obtained, and this will certainly be of significance.

Before the practitioner refers the patient for electrocardiographic examination with a view to finding whether coronary sclerosis or occlusion is present, he must, of course, have some clinical impression of its possible presence. In this respect a careful history is easily the most important factor in guiding the practitioner as to the significance of thoracic pain.

Typically the pain of angina of effort comes on with a definite amount of exercise, especially if taken after meals or in the cold air, causes the patient to stop, goes away quickly with rest, and subsequently tends to come on more readily, while the stabbing or "knife-like" pains of the anxious and flatulent dyspeptic, the pectoral pain which is brought on by putting tension on the *pectoralis minor* muscle, and the persistent submammary ache of the tired and anxious woman, are all usually easy of differentiation from the more serious condition. However, there are many occasions when uncertainty exists in the mind of the practitioner, and many more cases where it is present in that of the patient!

I believe that it is even more important to disabuse the patient's mind and free him from anxiety when no true cardiac damage is present than it is to diagnose coronary sclerosis which will, in any case, to some extent look after itself by limiting his activities.

In the presence of a completely negative clinical and special examination the cardiac neurotic may be reassured by the picture of a normal electrocardiogram which he can see in front of him.

But the practitioner must not be deluded into believing that a normal electrocardiogram necessarily excludes coronary sclerosis and angina of effort.

When the history is typical of angina of effort, diagnosis must be made even in the face of a negative clinical and electrocardiographic examination, for such is often the case. Sometimes, however, by means of exercise tests or allowing the patient to breathe a low concentration of oxygen, changes may occur in the electrocardiogram indicating ischæmia.

As serious complications have occurred as a result of exercising badly selected patients, I must urge that in order to protect the patient from the physician, and the physician from the relatives, great care must be taken in choosing patients for exercise tolerance tests. Some anginal patients who persist in providing normal electrocardiograms in spite of these efforts will show typical changes in the tracing if they are taken during a true attack of angina of effort.

Other changes in the myocardium due to hypertrophy and/or strain give rise to changes in the auricular and ventricular complexes; so in conditions such as hypertension, for example, when the strain falls on one ventricle more than the other, the electrocardiogram may indicate its effect.

These changes must not be confused with the changes brought about by alterations in the position of the heart, which frequently varies considerably with the height of the diaphragm.

In congenital heart disease the electrocardiogram is usually necessary, but in view of the frequency with which apparently simple congenital lesions are subsequently shown to be complex, the finer details of differentiation usually lie more within the ambit of the specialist than of the general practitioner.

In fact it is very frequently found necessary to resort to angiocardiography and cardiac catheterization before the riddle can be solved. When doubt exists whether one is dealing with a congenital dextrocardia of the innocent variety or with displacement of the heart to the right side by some extracardiac cause, the electrocardiogram, which shows an inversion of all waves in lead I, will give the answer.

During the course of an acute, or even a chronic, illness a suspicious friction rub or a rapid diminution in volume of the cardiac sounds together with an increased area of cardiac dulness may suggest the presence of pericarditis.

This may be confirmed by typical electrocardiographic changes. When elderly patients have been recumbent for long periods, or when they are recovering from the effects of surgical interference or coronary occlusion, the sudden onset of dyspnœa, and possibly of pain, must always raise the question of pulmonary embolism.

Hæmoptysis and local evidence of lung damage do not always come to our assistance, and the electrocardiogram itself may be normal, but if the electrocardiographic examination is repeated at intervals of a few hours it is quite likely that typical changes, which are often ephemeral, will be found. It must not be forgotten that trauma applied to the chest may cause myocardial damage even when the violence is not applied directly to the heart.

So in these cases when unexpected precordial pain, tachycardia or dyspnœa present, an electrocardiogram should be kept in mind. Many general conditions will give rise to changes in the electrocardiogram, for example, infections, intoxications, myxœdema, beriberi, anæmia *et cetera*, but it is only occasionally that the practitioner will need the aid of the instrument in this field where other investigations are usually of greater relative importance. However, there are times when cardiac symptoms appear to be out of proportion, and then instrumental aid must be sought. Chemical factors also affect the electrocardiogram, for example, the balance of potassium, calcium *et cetera*, or acid base changes in the blood, can cause changes which are sometimes typical and often gross.

What Call is there for the Electrocardiogram in Controlling Treatment?

As digitalis is still perhaps the most important drug in cardiac therapy, we must consider some of its effects. It slows conduction and depresses the *S-T* interval, which eventually slopes downwards to an inverted *T* wave. If given in toxic doses it causes ventricular premature contractions and may cause coupling, heart block and more dangerous complications. When the patient is distressed by vomiting and the practitioner is uncertain whether this is due to congestive cardiac failure or to digitalis, the electrocardiogram will decide.

In the treatment of auricular flutter with digitalis electrocardiographic control should be maintained because, if fibrillation appears, the digitalis should be stopped to await the return of normal rhythm. Similarly, when quinidine is used in auricular fibrillation, it should be watched lest I:I flutter should occur and the myocardium fail owing to the excessive speed.

During the course of coronary occlusion the appearance of any irregularity should be noted, and when its nature is in doubt it should be immediately determined by electrocardiogram so that treatment may be instituted if necessary.

Auricular fibrillation which does not subside spontaneously within twenty-four hours must be treated, while ventricular premature contractions suggest the use of quinidine or "Pronestyl" to prevent the development of ventricular paroxysmal tachycardia and ventricular fibrillation.

In acute rheumatism and infections a changing electrocardiogram indicates that the process is still active and is therefore an indication for a further rest in bed.

Aids to Prognosis.

In acute illnesses clinical evidence of cardiac involvement sometimes calls for an electrocardiogram; for example, in diphtheria there may be gross changes including varying degrees of block which indicate the gravity of the prognosis.

In acute nephritis cardiac manifestations sometimes are an outstanding feature, and the importance of this condition may warrant electrocardiographic investigation in order to follow the course of the damage. In coronary disease there is no accurate parallel between the degree of damage and the amount of electrocardiographic change, so on bundle branch block, for instance, no precise prognostic estimate can be based. However, in infarction multiple chest leads may to some degree indicate the extent of the infarct, which, when large, must enhance the gravity of the situation.

The onset of an abnormal rhythm in these cases is an adverse sign, especially when it causes tachycardia, while ventricular premature contractions when multifocal or occurring in groups are a potential danger.

Other unfavourable prognostic signs are a very low amplitude in leads 1, 2 and 3, extension of the infarct or the occurrence of a second one, as shown by serial tracings.

A patient with auricular paroxysmal tachycardia frequently has a normal heart, but the ventricular type usually indicates myocardial damage. In cases of hypertension and other conditions where there is extra work for the left ventricle, hypertrophy or strain causes the so-called left ventricular strain pattern, which is very definitely a sign of the progress of the condition.

Conclusion.

I have thus surveyed some of the more practical sides of electrocardiography from the practitioner's point of view. There should be no need to stress the point that a mere report on an electrocardiogram is not of much use alone, but the physician with a good clinical knowledge of his patient can use it to the best advantage along with his other evidence. Under such circumstances there are many occasions when the electrocardiogram will be of valuable assistance.

Summary.

Some of the factors influencing changes in the electrocardiogram have been mentioned.

The electrocardiograph records changes in rhythm, conduction and the condition of the myocardium.

It is of use in diagnosis, control of treatment, and prognosis. Two most useful roles are: (*a*) to reassure and help the cardiac neurotic; (*b*) to aid in the investigation of coronary artery disease and its complications.

On the whole, the electrocardiogram is of use only when viewed in correct perspective along with the other evidence so that the patient's condition may be considered as a whole.

E. F. GARTRELL,
Adelaide.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on December 13, 1951, at the Robert H. Todd Assembly Hall, British Medical Association House, 135 Macquarie Street, Sydney, DR. K. C. T. RAWLE, the President, in the chair.

Poliomyelitis and Murray Valley Encephalitis.

PROFESSOR SIR MACFARLANE BURNET read a paper entitled "Poliomyelitis and Murray Valley Encephalitis: A Comparison of Two Neurotropic Virus Diseases" (see page 169).

PROFESSOR H. K. WARD said that it was an occasion when Sir Macfarlane Burnet came to Sydney; it happened much too rarely. It was always a pleasure to be exposed to the workings of a fine mind and at the same time to hear an unrivalled exposition of its ideas. Professor Ward wished that Sir Macfarlane Burnet had had time to say a little more about parasitic pathways and how the neurotropic viruses reached the central nervous system. Attention had been drawn to the occurrence of localized paralysis following inoculation. The question of the prevention of poliomyelitis was one of great public importance. Sir Macfarlane Burnet was going to America in January, 1952, and would have an opportunity of speaking to workers in that field. Perhaps when he returned to Australia he would be able to report that there was a possibility of immunizing people against the Brunhilde strain of poliomyelitis virus. There were some faint hopes on the horizon that that event was not far off.

PROFESSOR LORIMER DODS asked if Sir Macfarlane Burnet felt that there was sufficient scientific justification for excluding from school for some traditional period those children who had been in close contact with a patient suffering from poliomyelitis.

DR. N. LEE asked whether there was any correlation between the incidence of Murray Valley encephalitis in visitors to the area and that in the inhabitants. He also wondered whether there would be any practical application in estimating the antibody titres in the serum of intending visitors to the area, and also in those of troops in the battle areas in Korea and Japan.

MR. N. F. STANLEY said that workers at Prince Henry Hospital were at present engaged on similar research. They were studying some of the problems associated with the epidemiology of poliomyelitis in New South Wales. This involved the identification of the strains of poliomyelitis present. Investigations had revealed many strains of Coxsackie virus as well as the poliomyelitis viruses. Interference phenomena between Coxsackie and poliomyelitis viruses were being studied.

DR. J. R. SHANNON said that he was Medical Officer of Health for the Hunter River district. He had a question to ask, but admitted that it might have a political bias. Sir Macfarlane Burnet had said that possibly the best method of controlling Murray Valley encephalitis would be by controlling the mosquito; but he had not said that he would urge that that should be done. Dr. Shannon said that those in his district lived on the estuary of a river, the population being about 262,000 people; they had had great success in controlling all types of mosquitoes. It was now almost impossible to be bitten on that estuary. No great cost had been involved; the method required a few teams and the employment of a fish, Gambusia. Dr. Shannon asked whether Sir Macfarlane Burnet would urge that in New South Wales the idea of mosquito eradication should be taken up with greater vigour than in the past, in view of the possible occurrence of encephalitis B.

DR. D. G. HAMILTON referred to the question of immunity. He said that he took it that infection with any one of the strains of virus gave immunity to that strain only. He asked what relation the Coxsackie virus bore to the Lansing, Brunhilde and Leon strains of poliomyelitis virus.

Sir Macfarlane Burnet, in reply to Professor Ward, who had asked about the spread of the virus from the alimentary tract to the central nervous system, said that the work of McCloskey in Victoria on the importance of prophylactic inoculations in the onset of paralytic disease opened up a difficult problem to which so far there was no satisfactory answer. Still, in the light of the work of Enders the view that the poliomyelitis virus was a strict neurotropic virus might have been too rigidly held. It was still orthodox to believe that the approach was via nerve paths outside and inside the central nervous system, but it was impossible to make any dogmatic statement about the localizing effect of prophylactic inoculations. In reply to Professor Dods, who had asked about the exclusion of child contacts from school, Sir Macfarlane Burnet said that the answer to that was very difficult; it was tied up with all sorts of human prejudices. Leaving all that aside, he could say that there was probably a certain level of invasiveness of the strain current at any particular time, above which one should exclude children from school, below which one should not. He had had long talks on the matter with Sabin and others in the United States. It was obviously necessary for a young child to develop immunity to poliomyelitis preferably before early school life, certainly before adult life. It seemed urgent that children should pick up poliomyelitis virus when non-invasive strains were current in the community. Sometimes it was possible to say that the poliomyelitis virus going round was non-invasive—for example, when there was one case a week of poliomyelitis in the metropolis, and not a steady trickle of cases. Other circumstances had prevailed in the 1937 epidemic in Victoria. Then cases occurred in a particular suburb in association with a particular school; it was possible to trace the infection from case to case or by way of one intermediate case. It was correct to stop all movement of children at such a time. But one had to ask at what stage the line was to be drawn. Sir Macfarlane Burnet did not think that there was any academic criterion; he would take the practical criterion that where poliomyelitis was prevalent enough to create considerable alarm, one was dealing with a highly invasive strain, and one could think of closing the schools. If the

disease was going on at a rate which the public would tolerate, one should think of immunization. A good case could be made for never closing the schools at all; however, he himself would make the differentiation he had given—an invasive strain associated with a high incidence of paralysis against a non-invasive strain. Dr. Lee had inquired about visitors to endemic regions of Murray Valley encephalitis. Sir Macfarlane Burnet, in reply, said that until they had had one more season's experience, he would be inclined to advise that they simply do nothing about the matters mentioned by Dr. Lee. The risk was extraordinarily low; in adults the risk of encephalitis following infection by mosquito was something less than one in 1000. It was not known what was going to occur; the reason why the disease had flared up in the previous year was not known. It might flare up again in the present season. If so, the investigators hoped to find the essential factor that allowed that unusual spill-over from the infective wild reservoir to humans. Another aspect of the question of visitors had been considered; they had compared the reactions of recent arrivals in the Mildura area as against those who had had a long residence. No difference in the proportion with antibody was found between the two groups. They had also investigated students of the University of Melbourne who had spent one year at the university's Mildura branch some time previously. The students had spent their time in Mildura mostly outside the hot summer months. Probably for this reason only one student gave a positive antibody result, and she was a resident of northern Victoria, and had probably been infected elsewhere. In reply to Dr. Shannon's question, Sir Macfarlane Burnet said that it was pertinent. He agreed with Dr. Shannon's point of view that where mosquitoes were an ordinary pest an attempt should be made to get rid of them. But great care was necessary at the present stage in advising control in order to get rid of Murray Valley encephalitis. One had first to decide which mosquito was involved, and get a picture of its life history, vulnerable aspects and so forth, before anything could be done. Until it was possible to isolate samples of virus from mosquitoes and define the species, he would not advise anti-mosquito measures. If any fresh outbreak of the disease occurred, there would be a demand for anti-mosquito measures, and then there would be an opportunity to look at the problem. But they did not have to contend with encephalitis always; they might find out what had been the particular factor in the previous year that caused the spill-over, and prevent it from operating again without anti-mosquito measures. In reply to Dr. Hamilton, who had inquired about strain immunity in poliomyelitis, Sir Macfarlane Burnet said that the Brunhilde type presented the only important problem. If they could obtain satisfactory immunization against it, they could forget the other two. The indications in America were that the two main strains and probably Leon passed through the community and conferred immunity at about the same rates. There had been instances in which antibody was produced against one but not against the other; for example, among the Eskimos there were antibodies against Lansing but not against Brunhilde. The practical problem was immunization against Brunhilde. Referring to the Coxsackie viruses, Sir Macfarlane Burnet said that he thought it a fair statement to say that there was no clear evidence that they had anything to do with poliomyelitis. He was open to correction on that point. Some authorities thought that their coexistence with poliomyelitis viruses increased the likelihood of infection; others wondered whether such coexistence might diminish the likelihood of infection. The Coxsackie viruses formed an extremely heterogeneous serological group which needed to be clarified. Two minor conditions—Bornholm disease and a particular type of pharyngitis—were thought to be related to the Coxsackie viruses. Sir Macfarlane Burnet concluded that with regard to poliomyelitis they should concentrate on the Brunhilde type of virus.

DR. EDGAR THOMSON, on being called upon to propose a vote of thanks to Sir Macfarlane Burnet, said that he had much pleasure in conveying to him the thanks of the Council of the New South Wales Branch of the British Medical Association, the members and the visitors. The address had been a rare treat. It was something that happened perhaps once in twenty years. Sir Macfarlane Burnet was a very busy man; he was always willing to visit other centres, he always gave of his very best, and whatever he said always gave his audience plenty to think about. He helped with many problems. One was tempted to hope that in Sydney the problem of virus infections would become so great that Sir Macfarlane Burnet would be tempted to spend some time there. One of his remarks had given his hearers great heart. Sometimes he (Dr. Thomson) had to talk to an audience of student nurses about the problem of prevention of disease and give them some elementary information about the problem of immunity. He had told them that when Johnny dropped his piece of bread and butter on the carpet, he should be made to pick it up and eat it. Sir Macfarlane Burnet had endorsed his remarks. Dr. Thomson then referred to the world-wide esteem in which Sir Macfarlane Burnet was held, and said that no other member of the medical profession in Australia was so widely known and respected. Those present felt sincere and deep gratitude to him for attending the meeting and giving them such an excellent address.

DR. A. W. HOLMES À COURT, on being called upon to second the vote of thanks, said that he could only repeat Dr. Thomson's remarks. Those present all felt that Sir Macfarlane Burnet's visits were too infrequent, but they realized that he belonged not to Australia, but to the world, so they must not be greedy. Dr. Holmes à Court said that he would like to hear what Sir Macfarlane Burnet had to say when he returned from America. He had given a masterly and lucid exposition of the subject, of enthralling interest to his hearers. They were grateful to him.

Sir Macfarlane Burnet, in reply, said that Dr. Anderson and his colleagues were continuing the investigation at full steam. If anyone had information about possible cases of Murray Valley encephalitis particularly in the next two or three months, he would be grateful to hear from him. At the Walter and Eliza Hall Institute they were anxious to establish the range over which it occurred in New South Wales, and it would be of the greatest help if information about any cases and specimens of serum could be obtained. They were anxious to get the situation clarified, and any help would be greatly appreciated.

A MEETING of the South Australian Branch of the British Medical Association was held on October 25, 1951, at the Verco Theatre, Institute of Medical and Veterinary Science, Adelaide, DR. R. L. THOROLD GRANT, the President, in the chair.

Tuberculosis and the General Practitioner.

DR. P. WOODRUFF read a paper entitled "Tuberculosis and the General Practitioner" (see page 186).

DR. A. C. SAVAGE, in opening the discussion, said that he had been particularly pleased to hear the stress laid on the public health aspect of the disease. The social benefits were now on a much better scale than when he had taken charge at Bedford Park twelve years earlier; at that time 17s. per week was deducted from the pension, 6s. 6d. being left to pay the rent and buy clothing and extras for the rest of the family. The question of B.C.G. vaccination was becoming increasingly important in the control of tuberculosis, as was illustrated by the amazing figures of the United States Army Health Department in Japan. The problem of control of patients in a sanatorium had been difficult in the past; probably 75% of patients discharged themselves voluntarily before they were medically fit to be returned to their homes. One patient had discharged himself because he was not permitted to take his rest period on the Brighton sands with his girl friend. Dr. Savage thought that it was not a great task for the general practitioner to perform a Mantoux test on all his private practice patients; those who yielded a positive reaction could be sent for X-ray examination.

DR. C. T. PIPER discussed the problem of adequately investigating patients in country areas, and also the difficulty of making patients suffering from primary pleurisy with effusion rest for long enough and continue to report back for observation.

DR. J. L. HAYWARD drew attention to the risks run by nurses in general hospitals, and to the relatively high incidence of tuberculosis in patients attending the ear, nose and throat department, the skin department *et cetera*—in other words, in those suffering from chronic ill health. Dr. Hayward also stressed the importance of B.C.G. vaccination in selected groups.

DR. D. R. WALLMAN pointed out that it was difficult for general practitioners to obtain fresh supplies of a 1:1000 dilution of old tuberculin. If it was easily available, they would use it more.

DR. F. L. WALL asked the earliest age at which patients could be given B.C.G. He said that it was a great advance that primary pleurisy with effusion could now be notified

as tuberculosis, so that the patients could qualify for the tuberculosis allowance.

DR. A. R. SOUTHWOOD commented on Dr. Woodruff's work as Director of Tuberculosis. He said that great changes had been made in recent years, in knowledge, facilities and techniques; but they must not forget the importance of better food, housing *et cetera*.

DR. R. MUNRO FORD said that PAS had to be used with streptomycin, and that dihydrostreptomycin was more toxic than streptomycin.

DR. R. C. ANGOVE said that the general practitioner could do much to allay the fear of tuberculosis among the general population, as it was no longer a fatal disease.

DR. J. R. MAGAREY asked for information concerning chemotherapy in surgical tuberculosis.

DR. H. HALLORAN commented on the vast improvement that had occurred in recent years. He said that there were now no really ill tuberculosis patients in his practice.

DR. M. W. MILLER asked for information concerning the patch test, in view of the difficulty of keeping fresh solutions for Mantoux testing.

Dr. Woodruff, in reply to Dr. Piper, said that at the Chest Clinic they received X-ray films and brief case histories from a number of country centres. The films were reported on by a radiologist, and a report was prepared by one of the chest physicians. Judging from the numbers of films submitted, he thought the service was appreciated. In addition, a State X-ray health survey unit had made one visit to most of the major towns of the State. Referring to the notification of pleural effusion, Dr. Woodruff said that he understood that the subject was shortly to be discussed by the National Health and Medical Research Council. It had been discussed by tuberculosis authorities. His own view was that unless some other policy was adopted throughout the country, they should continue to notify cases of primary pleural effusion as cases of tuberculosis. Dr. Hayward had drawn attention to the importance of chronic ill health in the elderly, and had emphasized the need to bear tuberculosis in mind in such cases. Dr. Woodruff said that he would add that that was particularly true in relation to elderly male patients, among whom morbidity and mortality rates from tuberculosis were strikingly higher than among females. Dr. Southwood had suggested that improved tuberculosis statistics might be due in large measure to general social betterment. Dr. Woodruff said that that was undoubtedly true; but he believed that those conditions had now improved so much, and tuberculosis rates had fallen so far, that the law of diminishing returns must operate. Further advances would require greater efforts on the part of medical practitioners. Dr. Wall had asked about the earliest age at which infants could be given B.C.G. vaccine. Dr. Woodruff said that some authorities—for example, Rosenthal in Chicago—gave it within the first few days of life. He himself believed that it should be given at one month, and that segregation of the child from the mother should be complete at least until Mantoux conversion had been demonstrated. In reply to Dr. Miller, who had asked about the reliability of the patch test, Dr. Woodruff said that, although he had had very little experience of its use, he thought that it was about equal to the Mantoux test when applied to young children with thin skins, but was less reliable when applied to adults. Dr. Woodruff said that he would examine for Dr. Wallman the possibility of supplying fresh dilutions of old tuberculin for use by general practitioners. Dr. Ford had drawn attention to two important points in relation to chemotherapy. Dr. Woodruff, in reply, said that he wished to stress again the fact that streptomycin should be used only in combination with PAS or thiosemicarbazone. In reply to Dr. Magarey's question about chemotherapy in surgical tuberculosis of non-pulmonary distribution, Dr. Woodruff recommended the use of streptomycin, one gramme per day, with PAS, 10 to 15 grammes per day, a commencement being made at least a week before operation. The administration of streptomycin should continue to a total of 50 grammes, and that of PAS for a period of three months. Dr. Woodruff said that it was heartening to hear Dr. Halloran say that really ill patients with tuberculosis had almost disappeared from general practice. It was not so long since the senior clinical teachers used to tell students of the days when there was always a ward full of typhoid patients in the major general hospitals. It would require a concerted effort to make tuberculosis as rare as typhoid had become.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

THE HOSPITAL.[1]

[*The Colonist*, September 28, 1837.]

For some time past, in consequence of gross neglect and mismanagement on the part of certain individuals both at home and in this colony a series of disputes have occurred amongst the officers of the Medical Department. Hitherto, although acquainted with the whole of the circumstances, we have abstained from offering any remarks on the subject, because we were induced to hope that justice would be administered to all parties, and that the department would be restored to its wonted order and regularity. As, however, there does not appear to be any likelihood of bringing the matter to an amicable adjustment, we are reluctantly compelled to introduce it to the notice of our readers. And this we do not from any personal consideration, but solely with a view of procuring a favorable hearing of Mr Mitchell's[2] case, and of restoring that valued officer to his public duties. It appears that about 18 months ago, owing to some egregious mistake on the part of the Home Government, a Mr Thomson was sent out to take charge of the Colonial Hospitals, and that he, inflated with "the insolence of office", issued various orders, which gave great dissatisfaction to several of the officers under him. Amongst the rest, Mr Mitchell, feeling himself aggrieved by the new regulations, was induced to bring sundry charges against the Head of his Department, but, being unable to make out his case to the satisfaction of the Court, he received a gentle reprimand from his Excellency the Governor. The ill success of this appeal naturally led to a renewal of hostilities on the part of the Deputy Inspector, who, in his turn, endeavoured to make out charges against Mr Mitchell. Without entering upon the subject at any length on the present occasion we merely mention, for the information of our readers, that Mr MITCHELL in his lost encounter with Mr Thomson, conceiving that he had been unfairly treated by the Military Court, refused to submit to the decision of a similar tribunal and referred his case to the Governor himself. The result is at present unknown, but, as it will transpire in the course of a few days, we shall be enabled to lay the particulars before the public in our next. Knowing that Mr Mitchell is an able and efficient officer, and that for a number of years he has performed his duties with credit to himself and satisfaction to the community, we feel deeply interested in his welfare and sincerely hope that he may triumph over his opponents.

Correspondence.

A CASE OF DEATH FOLLOWING SECTION, LIGATION AND INJECTION OF VARICOSE VEINS.

SIR: I have followed the correspondence since Dr. A. E. Coates reported his case (THE MEDICAL JOURNAL OF AUSTRALIA, September 22, 1951). I have devoted the last eighteen months almost solely to the private treatment of varicose veins and now have some confirmed views on some of the causes of complications from the above procedure. I have learned from my mistakes and do not wish them to occur again.

It is difficult to support Mr. V. M. Coppleson in his views on the satisfactory results and lack of complications from section, ligation and injection of varicose veins from the number of doctors who are unwilling to subject themselves to the above procedure.

Dr. C. H. Wickham Lawes is to be commended in bringing to our notice again that full strength "Ethamolin" is not necessary.

[1] From the original in the Mitchell Library, Sydney.

[2] James Mitchell, assistant surgeon at the General Hospital, Sydney, 1824, senior surgeon, 1825-1837. He was the father of David Scott Mitchell, the donor of the Mitchell collection to the New South Wales Public Library.

During the last few months I have been ligating the saphenous vein in the *fossa ovalis* and any major junctions or "blow outs" until the internal malleolus is reached, using local anæsthesia with the utmost comfort to the patient and easy operating conditions. The patients leave the operating room and travel home by train, car or bus and have little discomfort until two hours later, by which time they have got over the idea of being an invalid. Although operating may take two or more hours when two legs are operated on, it is appreciated by the patients, who are fit for light work in two days and return in ten days to have the stitches removed, when they are cured in the majority of cases, not needing to return for any injections. In extremely bad cases they are fit for hard work in three weeks.

I cannot agree with Dr. Mervyn Hall that where there is a large saphenous tree there are ever no anastomosing veins. It is instructive to accurately dissect bunches of varicose veins and see how impossible it is to be sure where full strength "Ethamolin" will finish. Most of the pain and swelling of the leg and inability to return to work comes from what is injected into the veins and not from the number of incisions in the leg.

Yours, etc.,

229 Hunter Street, Newcastle, January 27, 1952. J. N. R. STEPHEN.

SIR: As there has been much discussion of late about the effects of injecting "Ethamolin" as a sclerosant in varicose veins, it was thought that the following report might be of interest.

In November and December, 1944, A.R.M. was given weekly injections into veins in his right leg. Seven injections, each of two cubic centimetres of "Ethamolin", were given, with considerable benefit.

The patient returned on January 4, 1952, with marked recanalization, and a fresh series of injections was begun, the same dosage being used. No untoward reaction occurred after the first three treatments.

At 10 a.m. to-day a further injection of two cubic centimetres of "Ethamolin" was given. Within a minute or two the patient said he felt "queer". On inquiry he said he felt as if he were going up and down in a lift. Pulse rate was 96 per minute, colour unchanged. Seven minims of 1:1000 adrenaline solution were given hypodermically. He next complained of feeling itchy round the neck, where urticarial weals were noted. "Phenergan Elixir", one drachm (five milligrammes), was given. It was some fifteen minutes before the patient could stand up and take a seat in the waiting-room and about two hours before he was able to go home.

Yours, etc.,

7 Pittwater Road, Gladesville, January 24, 1952. A. W. J. BULTEAU.

APPENDICITIS IN CHILDHOOD.

SIR: I feel I must reply to Dr. Smyth's letter (THE MEDICAL JOURNAL OF AUSTRALIA, January 19, 1952) criticizing certain statements in my article on "Appendicitis in Childhood" (THE MEDICAL JOURNAL OF AUSTRALIA, December 15, 1951).

Firstly, Dr. Smyth takes from its context the second part of the following sentence and honours it with a preliminary capital as though it were a complete statement standing alone: "It is also necessary to state that our figure for cases of perforation really includes only those of perforation plus drainage; an unknown percentage of cases classified as 'acute without perforation' were in fact early cases of perforation in which there was so little spread of pus that the surgeon closed the abdomen without drainage." Surely that statement is capable of being understood, even in court. Perhaps I should have added the further explanation that I did not personally check the whole of the 2206 case histories for the four-year period to find out how many had an early rupture, but had been closed without drainage.

On the other hand, figures quoted by me in the more detailed classification of cases from July, 1948, to June, 1949, are all accurate, to the extent that each case history was personally investigated. In that year there were 27 cases of perforation in a total of 403 operations, an incidence of 6·7%.

The point, which I thought I had made clearly enough for an intelligent reader to understand, was that our four-year figure (4·6% of cases) covered only cases of perforation plus drainage, and was not strictly comparable with the higher percentages in other series which presumably included all cases which had perforation of the appendix.

Secondly, Dr. Smyth criticizes somewhat harshly the sentence: "Seldom can one expect the pathologist to substantiate a diagnosis of 'subacute' or 'chronic' appendicitis. These terms are used in a clinical, everyday, sense rather than in a true pathological sense." I make no apology for these statements, as I believe them to be true, having taken the trouble to send to the pathologist many appendices from cases which were admitted to hospital with such a diagnosis. If Dr. Smyth would take the trouble to discuss this point with a pathologist I am sure he would be told that few appendices show evidence of genuine subacute inflammation, and very few show evidence of chronic inflammation, although they may show evidence, in the form of fibrosis, of a previous severe acute inflammation.

It is my belief that clinicians should only use the term "subacute appendicitis" in cases which have had continuous symptoms and signs suggesting appendicitis for a period of several weeks and not in cases which should correctly be described as "mild, acute appendicitis". It is also my belief that clinicians should abandon the use of the term "chronic appendicitis" in favour of the term "recurrent mild appendicitis" or "recurrent appendical colic".

Dr. Smyth states: "In the past a criterion of correct diagnosis was that it could be confirmed by the pathologist." It is precisely for that reason that I made the statement to which Dr. Smyth takes exception. Perhaps the additional statements now made will be considered to constitute the "new and better definition" which Dr. Smyth would like. In conclusion I may add that, throughout my article, I used the terms "subacute" and "chronic" appendicitis in inverted commas; perhaps I am being pedantic, but I do believe we should always attempt to use medical terms which are as accurate as present knowledge will allow.

Yours, etc.,

123 Miller Street, North Sydney, New South Wales. January 21, 1952. EDWARD S. STUCKEY.

Post-Graduate Work.

THE MELBOURNE PERMANENT POST-GRADUATE COMMITTEE.

PROGRAMME FOR MARCH, 1952.

Classes for Higher Degrees and Diplomas, Part I.

Anatomy.

Anatomy classes which commenced in February will continue on Monday and Wednesday afternoons at 2 o'clock.

Pathology and Physiology.

On Monday and Wednesday afternoons, at 1.45 o'clock, commencing on March 17, pathology lectures will be held in the Pathology Department, University of Melbourne, for candidates for Part I M.D. and Part II of M.S. and diplomas, followed by pathology practical demonstrations in the pathology laboratory; at 3.45 p.m., commencing on March 3, physiology lectures will be held in the Physiology Department for candidates for Part I M.D., M.S., D.A., D.G.O., D.O., D.L.O., D.P.M. and D.D.R.

The first half of the pathology course is suitable for Part II of various diplomas and M.S., and will be supplemented where required by special lectures and demonstrations suitable for the degree or diploma concerned.

Physics.

On Thursdays, at 4.30 p.m., commencing on March 6, physics lectures will be held in the X-ray and Radium Laboratory for candidates for Part I D.D.R. and D.T.R.

Fees.

The fees are £31 10s. (total) for courses for Part I of a degree or diploma and £15 15s. for each subject taken separately.

Courses Suitable for Candidates for M.D. II—M.R.A.C.P.

Pædiatrics.

Pædiatrics, under the direction of Dr. M. L. Powell, will be demonstrated at 2 p.m. on March 18, 21, 25, 28, April 1, 4.

Course in Poliomyelitis.

The Melbourne Permanent Post-Graduate Committee will arrange a course of instruction in the diagnosis, early treatment and after-care of poliomyelitis at Fairfield Hospital on Saturday, March 29. The meeting will commence at 11 a.m. and continue until 4 p.m. Lunch will be available at the hospital. A detailed programme can be obtained on application to the Post-Graduate Committee.

Country Courses.

Mooroopna.

A week-end course will be held at Mooroopna on March 1 and 2. The programme is as follows:

Saturday, March 1: 2.30 p.m., Dr. J. McB. White, "Treatment of Common Diseases of the Eye"; 4 p.m., Dr. W. J. Rawlings, "Indications for Operative Interference during Labour".

Sunday, March 2: 10 a.m., Dr. C. J. O. Brown, "Selection of Cases for Cardio-Thoracic Surgery"; 11 a.m., Dr. W. E. King, "Thyrotoxicosis, its Diagnosis and Management".

The fee for this course is £2 2s., or 10s. 6d. per demonstration. Enrolments should be made with the Secretary of the Subdivision, Dr. B. R. Schloeffel, Maud Street, Shepparton (telephone 67).

Flinders Naval Depot.

Dr. K. Bradley will lecture on "Pain Syndrome" at the Flinders Naval Depot on March 12 at 2.30 p.m., by arrangement with the Royal Australian Navy.

Enrolments.

Enrolments for the courses in the metropolitan area should be made with the Post-Graduate Committee not later than two weeks before the commencement of the course. Fees are payable in advance.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Clinical Meeting at Balmoral Naval Hospital.

The Post-Graduate Committee in Medicine in the University of Sydney announces that a clinical meeting will be held at the Balmoral Naval Hospital on Tuesday, February 12, 1952, at 2 p.m., when Dr. I. Douglas Miller will speak on "Head Injuries". Clinical cases will be shown at 4 p.m., after afternoon tea. All members of the medical profession are cordially invited to attend.

Obituary.

MAY HANNAH HARRIS.

We are indebted to Dr. Curzon W. Furner for the following appreciation of the late Dr. May Hannah Harris.

With the death of Dr. May Harris on October 16, 1951, Newcastle lost its senior practitioner and New South Wales one of its first two medical women. She was a member of possibly a record medical family which produced seven doctors, who at one stage, and for a considerable period, were in practice at the one time. After her preliminary arts course in Sydney she studied at Edinburgh, Dublin and London, where she graduated in 1895. On returning to Australia, she went into practice with her father, the late Dr. John Harris.

When workers' compensation became law, Dr. John Harris was appointed medical referee for the Mine Owners' Insurance. This work necessitated not only the examination of all injured miners who came to Newcastle, but also weekly trips to all mines insured by this company on the coal fields. Further to this, there was a considerable amount of court work arising from the many claims for compensation.

During his long illness, Dr. May Harris carried on her father's work, and after his death in 1923 was appointed in his place. The work grew in extent and responsibility, and the organization which her father founded was faithfully and most ably carried on and developed. She was keenly interested in mine gases and became so well known for her expert knowledge in this aspect that no judge would question her authority in court. She was instrumental in perfecting the formula of "Carbogen", and it was by her repeated efforts that the "Proto" apparatus was adapted. Both these innovations were tested and proved life-saving assets in the famous Bellbird disaster. Her greatest interest was undoubtedly "dust", and particularly as related to the coal mines. It was mainly her research *plus* her persistent requests to the coal authorities to accept the fact that its control was of vital importance to the industry that pneumonokoniosis was recognized in Australia long before anything was done about it in England.

Dr. May had a positive personality. She was an excellent medical witness, for she had complete confidence in her own considered opinion and was inflexible under cross-examination. She was forthright in her speech and straight in her manner. To say that all the miners liked her would be untrue, but they did respect her. Very few braved the consequences of her wrath by attempting to malinger. In her private life she was retiring and reticent, and apart from extensive reading she indulged in a few hobbies. She was artistic and could draw extremely well. She was also fond of woodwork and spent much time in her workshop. She was kind and generous and helped many people in their necessity, but always under the pledge of absolute silence.

Dr. May retired from active practice ten years ago because of advancing arthritis, which she bore stoically and with the help of her favourite literature, archæology, history and detective thrillers, not forgetting the incessant cigarettes.

Dr. May would not desire that anyone mourn her death. Her friends knew that her one desire was to leave us when the days of usefulness had passed. She believed that life was not worth while and only a hindrance to others when the powers of activity and mobility were gone. We did not believe this ourselves, for with her mind so clear and alert even to the last few days, one always felt the better for having visited her. She was a great character, perhaps difficult to understand fully, but when she was understood, and furthermore when one was accepted by her as a friend, she was indeed inspiring, and one felt very glad of her friendship. It was a privilege to know her.

> Sunset and evening star are one clear call for me,
> And may there be no moaning at the bar
> When I put out to sea.

Dr. Ethel Byrne writes: Dr. May Harris, who died at the age of seventy-nine years in October, 1951, was the last of her medical generation in Newcastle and the district. She graduated in London in 1895, and on returning to Australia went into practice with her father, the late Dr. John Harris. They carried on a busy general practice, which extended over a vast area, for many years, depending for transport on horses and horse-drawn vehicles. Some of Dr. May's stories of harnessing up in the middle of the night to attend a confinement in a miner's cottage five or ten miles distant are epics of medical practice of the days when doctors had only their own skill and knowledge to combat illness of all kinds. When Dr. John Harris retired from general practice and became medical officer for the Mine Owners' Insurance. Dr. May carried on the practice unaided, winning the respect and affection of a large section of the community. One heard from many sources of her kindness and generosity throughout the district, and there was great regret among her patients when, on the death of Dr. John Harris, she agreed to take over his insurance duties. In her capacity as medical officer to the mining insurance interests she became a recognized authority on mine gases, pneumonokoniosis and other industrial conditions. After her retirement in 1951, she still retained her interests in mining conditions and could always be relied on to provide any literature relevant to a case under discussion. In the last three years of her life, she was practically an invalid, but she still kept herself conversant with current medical literature and was glad to discuss any problem one might bring. As relaxation, she read endless detective stories. Dr. May Harris was one of a big family of doctors. Her father and two brothers predeceased her, one brother is in practice in Newcastle, and there are others in various parts of New South Wales.

GEORGE AUGUSTUS HARDWICKE.

We regret to announce the death of Dr. George Augustus Hardwicke, which occurred on January 28, 1952, at Sydney.

JAMES MANNING HAIR.

We regret to announce the death of Dr. James Manning Hair, which occurred on January 26, 1952, at Sydney.

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 5, of January 4, 1952.

NAVAL FORCES OF THE COMMONWEALTH.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Appointment.—John Anthony Bloomfield is appointed Surgeon Lieutenant (for short service), dated 2nd April, 1951.

Citizen Naval Forces.

Royal Australian Naval Reserve.

To be Surgeon Commander.—Surgeon Lieutenant-Commander Clifford Kelvin Churches, M.B., M.S., D.G.O. (Melb.), F.R.C.A.S.—(Ex. Min. No. 3—Approved 14th January, 1952.)

AUSTRALIAN MILITARY FORCES.

Australian Regular Army.

Royal Australian Army Medical Corps.—To be Major, 27th September, 1951, with a Short Service Commission for a period of four years: 2/40110 Dudley Carlyle Cook (Major). To be Captain, 9th March, 1951, with a Short Service Commission for a period of two years: 2/40109 Leslie Leonard Nordstrom (Captain).

Royal Austalian Army Medical Corps.

VX700128 Captain (Temporary Major) R. G. Webb relinquishes the temporary rank of Major and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps) (3rd Military District), 6th February, 1951.

VFX700115 Captain M. Howson is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (3rd Military District), 19th October, 1951.

To be Captains (provisionally), 9th November, 1951.—VX700277 Kenneth Norman McNicol, VX700228 John Harry Leyden, VX700230 Joseph Stanislaus Crosbie and VX700229 Bryan Craig Aekins Stratford.

Citizen Military Forces.

Northern Command: First Military District.

Royal Australian Army Medical Corps.—1/32022 Captain K. H. Hooper is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (1st Military District), 16th October, 1951.

Southern Command: Third Military District.

Royal Australian Army Medical Corps.—3/63892 Captain J. W. Kent is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (3rd Military District), 1st October, 1951.

3/101015 Captain (provisionally) J. L. Stubbe relinquishes the provisional rank of Captain and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (3rd Military District) with the rank of Honorary Captain, 18th October, 1951.

Western Command: Fifth Military District.

Royal Australian Army Medical Corps.—The resignation of 5/32061 Major (Honorary Lieutenant-Colonel) J. M. M. Drew, O.B.E., of his commission is accepted, 1st September, 1951.

Reserve Citizen Military Forces.

Royal Army Medical Corps.

2nd Military District.—The resignation of Major A. O. Davy of his commission is accepted, 8th November, 1951. *To be Honorary Captain, 31st October,* 1951.—John Raftos.

6th Military District.—To be Honorary Captain, 31st October, 1951.—Ernst Fabian.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED JANUARY 12, 1952.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia[3]
Acute Rheumatism	..	..	..	..	..	..	..	..	..
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	1	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	..	..	17(16)	..	..	..	..	..	17
Diphtheria	7	2(1)	4(2)	..	3(3)	..	..	..	16
Dysentery (Bacillary)	..	..	4(4)	..	..	..	..	2	6
Encephalitis	..	3	..	..	..	..	..	..	3
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	..	..	..	..	9(6)	..	..	..	9
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	1	..	..	..	..	..	1
Leptospirosis	..	..	1	..	..	..	..	..	1
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	7(6)	4(2)	..	..	2(1)	..	..	..	13
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	14(5)	..	7(5)	24(16)	3(1)	1	..	..	49
Puerperal Fever	..	..	1	..	..	..	..	..	1
Rubella	..	6(6)	..	..	3(3)	..	..	..	9
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	11(5)	10(8)	..	4(1)	1(1)	3(2)	..	..	29
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	2(1)	3	..	..	..	..	..	5
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	21(13)	18(9)	10(3)	6(4)	16(10)	5(2)	..	1	77
Typhoid Fever	..	..	2	..	..	..	..	..	2
Typhus (Flea-, Mite- and Tick-borne)	..	..	2	..	..	..	..	..	2
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

[2] Figures not available.

[3] Figures incomplete owing to absence of returns from the Northern Territory.

Retired List.

The following officers are placed upon the Retired List within Military Districts and on the dates as shown, with permission to retain their rank and wear the prescribed uniform:

5th Military District.—Lieutenant-Colonels (Honorary Colonels) J. R. Donaldson, E.D., and E. J. T. Thompson, M.C., E.D., Major H. J. Bowra, Captain H. B. Kershaw and Honorary Captain H. E. Stevens, 7th November, 1951.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force: Medical Branch.

Thomas Fraser Sandeman (035954) is appointed to a short-service commission, on probation for a period of twelve months, 9th October, 1951, with the rank of Flight Lieutenant.

No. A16225 Warrant Officer Ronald Vivian Eedy (016225) is transferred from the Active Citizen Air Force and appointed to a short-service commission, on probation for a period of twelve months, 8th October, 1951, with the rank of Flying Officer.

The resignations of the following officers are accepted: Flight Lieutenant P. Grattan-Smith (021962), 22nd October, 1951, Flying Officer R. E. Robins (012652), 20th November, 1951.

Air Force Reserve: Medical Branch.

The following officers are placed on the Retired List, 1st September, 1951:—Wing Commanders (Temporary Group Captains) G. C. Willcocks, O.B.E., M.C. (1161), H. R. G. Poate, M.V.O. (1162); Squadron Leader (Temporary Wing Commander) E. O. Marks (1166); Flight Lieutenants (Temporary Wing Commanders) H. W. Savige (2077), E. M. Fisher (2056); (Temporary Squadron Leader) G. Finlay, O.B.E. (2617).

The following are appointed to commissions with the rank indicated:—(Flight Lieutenant (Temporary Squadron Leader)) John Henderson Bolton (257837), 27th July, 1951; (Flight Lieutenant) Charles Arthur Holmes (267919), 20th February, 1951, Edward Lucas Dunn (277684), 18th June, 1951, Ernest Henry Strugnell Mancy (267838), 6th July, 1951.

Research.

BRITISH COMMONWEALTH INDEX OF SCIENTIFIC TRANSLATIONS.

THE British Commonwealth of Nations Scientific Liaison Offices in London have started a Commonwealth index of scientific translations. Each of the Commonwealth countries has or will have an agency which is to maintain a central index of translations into English or published scientific papers, reports and journal articles which have been made by various organizations within the Commonwealth. Each agency collects details of translations made by the organizations in its own country that are cooperating in the scheme; the information is then passed on to the British Commonwealth of Nations Scientific Liaison Offices, London, where index cards are prepared and sent out to the agencies within the Commonwealth.

The Australian agency for this index is the Information Service, Commonwealth Scientific and Industrial Research Organization, 314 Albert Street, East Melbourne, C.2, Victoria.

Persons in Australia wishing to know if a certain translation is available should apply to the Senior Information Officer at the above address.

Corrigendum.

IN the issue of January 12, 1952, under the heading of "Honours" it was stated that Major-General Samuel Roy Burston had been created a Knight Bachelor. General Burston was, in fact, created a Knight Commander of the Most Excellent Order of the British Empire in the Military Division. We regret this error.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Low, Bruce, M.B., B.S., 1951 (Univ. Sydney), Royal North Shore Hospital, Crow's Nest.

Roach, Trevor Hunter, M.B., B.S., 1950 (Univ. Sydney), 1 Tennyson Crescent, Forrest, Canberra.

Diary for the Month.

FEB. 12.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
FEB. 19.—New South Wales Branch, B.M.A.: Medical Politics Committee.
FEB. 22.—Queensland Branch, B.M.A.: Council Meeting.
FEB. 26.—New South Wales Branch, B.M.A.: Ethics Committee.
FEB. 28.—South Australian Branch, B.M.A.: Scientific Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

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